Digitized by the Internet Archive
in 2018 with funding from
University of Toronto

https://archive.org/details/medjournalDec1936uoft

CONTENTS.

	Page
EDITORIAL	37
AN APPROACH TO ANATOMY—By J. C. B. Grant, M.B.	40
VARICOSE VEINS AND LYMPH STASIS—By John Harold Couch, M.B.	43
FRACTURES OF THE MANDIBLE—By Stuart Gordon, M.S. (Tor.)	46
APHASIA—By H. H. HYLAND, M.D.	49
DISSEMINATED SCLEROSIS—By I. J. Tepperman, B.A., '37	53
EPILEPSY—ITS CAUSES—By Anne Keyl, B.Sc., '37	60
EMPYAEMA: A REVIEW OF ITS ETIOLOGY, DIAGNOSIS AND TREATMENT—By A. D. Rice, B.A.	63
LEAD POISONING IN THE AUTOMOBILE INDUSTRY, PART II.—By Alastair Macdonald, '37	70
THE LEUKAEMIAS—By W. I. BRESLIN, '38	76
THE FOETAL SKULL—By Clement McCulloch, '39	81
FEATURE SECTION—	
NOTES OF A STUDENT ONE HUNDRED YEARS AGO—By E. Stanley Ryerson, M.D.	84
A SHORT VISIT TO HUDSON STUCK MEMORIAL HOSPITAL, FORT YUKON, ALASKA—By J. Wallace McNichol, '37	87

THE UNIVERSITY OF TORONTO MEDICAL JOURNAL

(An Undergraduate Publication)

This Journal is published six times a year, November to April, by the Medical Society of the University of Toronto, to serve as a medium for student expression and to bring to the student recent articles of scientific interest.

ADVISORY STAFF.
Prof. E. A. Linell, M.B., Ch.B., M.D. Manc.
J. Harold Couch, M.A., M.B., F.R.C.S. Edin., F.R.C.S. (C.).
H. H. Hyland, M.D., M.R.C.P. LOND., F.R.C.P. (C.).
D. Y. Solandt, M.A., M.D.
Prof. G. F. Marrian, D.Sc., Lond.

EDITOR-IN-CHIEF
Hubert Hayward, B.A., '37.

ASSOCIATE EDITOR — W. G. Bigelow, B.A., '38.
FEATURE EDITOR — J. P. Fletcher, M.A., '37.

ASSISTANT EDITORS
R. A. Mustard, M.A., '38. J. H. Belton, '39.
K. C. Greer, B. & M., '37.

WOMEN'S EDITOR — Miss J. A. McGeachy, '37.
MANAGING EDITOR — R. T. Gates, '39.

CIRCULATION STAFF
Manager—A. W Sturgeon, '37.

Assistants—
B. T. Rogers, 37. R. I. Probert, '40.
B. W. Vale, '38. V. V. Marinelli, '41.
W. O. H. Perry, '39. J. A. Hooper, '42.

Address all communications to Room 107, Anatomy Building.

Vol. XIV Toronto, December, 1936 No. 2

It is worth remarking that of the 6,209 hours of study prescribed in six years of the medical course, 120 only are devoted to physical training. This represents less than 2% of the total, a value substantially reduced when we consider that very few attend more than 80% of these periods and that 50% of that time is spent in preparing for exercises and for resumption of studies in civilian garb.

Average figures taken from the books of business endeavours indicate that 5% or so of gross receipts are set aside as a Sinking Fund, to cover depreciation and to maintain the proposition in as fine condition throughout its existence as it was at the founding.

An education in medicine represents a substantial investment valued at some $5,000 in cash, six years or more of the life of the finest class of manhood and womanhood in our land at the most productive period of their lives. It represents a loss of $5,000 and upwards in salaries over this period with little increase, if any, in lucrative capabalities—and yet less than 2% of time and much less of money is allotted to the maintenance of this adventure.

Drawing a lesson from the many the business world around us has to teach, where the cry of stockholders

and executives makes it imperative that business be run for profit, does it not seem that this student body and its foster parents are guiding "the ship of state" through perilous waters without due regard for evident signs of shoals? Do not so many of us continue to drain our reserves and to keep ourselves at a low ebb of physical and mental efficiency? Added to this is the fact that more advanced students are not only robbed of opportunities for the cultivation of healthful habits but are exposed to the hazard of acquired disease. Has the economic depression through which we have passed taught business much? We doubt it. And will we learn? We hope.

Students of medicine often find difficulty in grasping the true significance of the subject matter presented. From their acquaintance with other walks of life the wisdom in the advice, Rest, Reassurance and Physical Recreation, remains only as words. They have failed to turn facts into concepts and to realize the universality of these. Given the opportunity to devote more time to physical recreation, how few are willing. But of those who are willing, how slight their opportunities are, for to play games time must be stolen from hours of instruction so urgently needed. With the present set-up few can excel in games and study too. To them we raise our hats. The average student is neither a sportsman nor a scholar. Dulled by the heavy going, devoid of a vigorous constitution, without the exhilaration which only physical recreation can give, he limps through his undergraduate days to find solace in the discussion of future school years with his elders in study.

That students are interested in this problem stands the fact that at a meeting of the House Committee of Hart House last spring a resolution was passed, submitted to and supported by the Athletic Directorate, to the effect that gymnasia and swimming pool at Hart House be available in the evenings for the use of students in the clinical years in Medicine, who by interest in their work, prescribed duties and location of site of study find it impossible to use these during the school-day. It is unfortunate that the Board of Governors were unable to see their way clear to make this law, in spite of the fact that the upper years in Medicine contribute some $4,000 towards the upkeep of the House and are unable to utilize even a fraction of this—real philanthropists preparing by practice for the life of philanthropy that lies ahead.

We would appreciate that the staff and Board of Governors in whose hands an undergraduate's welfare lies, should see fit to supervise more fully not only his mental progress but the investment entrusted to them. We feel sure the student body would support a plan for the inclusion of physical training periods in the curriculum of all the years.

Open Meetings.

Those who were fortunate in having time and desire to attend the last open meeting of the Medical Society, were well rewarded by Dr. E. S. Ryerson. May we publicly acknowledge the efforts of Mr. W. I. Breslin for arranging such a gathering. Mr. Breslin has another treat in store. Arrangements are being completed for the next open meeting to take place on or about Jan. 14, 1937. Dr. Morris Fishbein, editor of the Journal of the American Medical

Association and author of some celebrated volumes—just to mention "One Thousand Million Guinea Pigs"—has consented to address the students of medicine upon "Fads and Quackery in Medicine." Need we say more?

In Defence of the General Practitioner.

The Editor,
University of Toronto, Medical Journal.

Dear Sir,—

Recently it has seemed to me that the general practitioner is receiving more criticism than he deserves. I should like to take up the cudgels in his behalf.

The medical student has a point of vantage in the estimation of the personnel of the medical profession. On the one hand he sees with a more critical eye than the layman, the activity of the general practitioner coming to a case in the home, whilst on the other he sees a variety of the specialists in the profession as they teach in the medical school and hospital wards. At times the academic meal set forth by the specialist, digests but poorly. It is too highly peppered with such remarks as, "A general practitioner (good man too!) saw this patient and failed to diagnose the condition"; or again: "A man outside saw this case, several times, and failed to recognize . . . etc., etc."

Perhaps the word "outside" is the most vicious of all. Outside indeed! These outsiders in the view of the public are very much on the inside, for without them the specialist would not exist. They form an essential part of the present medical system; they serve hard and long; they do not deserve derision.

Of course, they make mistakes. So do the best in any field. One well-known internist at Toronto is fond of remarking that the very best men in law or medicine are only correct in 80% of their efforts. If 80% precision can be reached only with:

(a) Large hospital facilities for laboratory, and cheap repeated X-rays.
(b) Expert advice from other fields.
(c) Extra time for consideration of a single problem—

surely the general practitioner can be excused for some errors! Furthermore, by the time the patient reaches the specialist the distance between existence and death has been narrowed, making it so much the easier to ascertain what will be the manner of death. In other words, the specialist is provided with hind-sight whereas the general practitioner must use fore-sight. He serves as the sorter of all the variety of cases and makes the work of the specialist easier. The latter should be grateful for this laborious service.

Having reached this attitude, one must consider what can be done to aid the general practitioner. The traveling clinics, such a scheme as that indicated by Mr. Kerr's article of the first number of this Journal for 1936 are steps in the right direction. We must guard the relation of the general practitioner and his families because that relation is the best for them. Therefore, any scheme for state medicine must recognize the family physician as the foundation-stone on which to build a superstructure of clinics staffed by intelligent, sympathetic specialists who will render service and avoid damaging criticism of the foundation on which they stand.

Caram.

AN APPROACH TO ANATOMY

By J. C. B. GRANT, M.C., M.B., Ch.B., F.R.C.S. (EDIN.),
Professor of Anatomy, University of Toronto.

The word Anatomy is derived from a Greek word, meaning literally "to cut up." The word Dissection is derived from the Latin dis-secare, "to cut apart." The prefix dis- is the same as that found in dis-sociate, dis-seminate and dis-locate. Many who practice dissection impose upon themselves a perpetual handicap by deliberately suffering removable obstacles in their paths. The sporting spirit seems to compel them to run a hurdle race in competition with others who have entered for a flat race; nor are they dissuaded by the spoken word or by the knowledge that the race is to be swift.

This article may suggest to junior students of Anatomy methods of works and lines of thought.

The two most formidable obstacles the student appears to delight to encounter are a neglected part and a blunt knife.

The Neglected Part.—Before the dissector assumes responsibility for the part, the body has been embalmed with antiseptics (including carbolic acid), hardening fluids (including spirit and formalin), hygroscopic agents (probably glycerine and potassium acetate) and salts; and the arteries have been filled with a coloured mass (probably starch, red lead and glue) to render them conspicuous. Intelligent care must be taken to keep the parts allotted for dissection in the condition in which they are received—firm and moist, but not sodden: a part that has been allowed to dry cannot be restored to its former state. The The part that should be surrounded with wet cotton waste held in place with a bandage; and the whole should be enveloped in a waterproof sac. Not even for a moment should more of a part than necessary be exposed. Parts should be unwrapped and refreshed periodically. Ignoring holes in the waterproof sac and letting the ends of cloths project beyond the mouth of the sac, so that they act as lamp-wicks, promoting loss of fluid, is treating the part with indifference.

Knives.—Two knives are required by every dissector. One is for rough work, such as cutting skin and tendons. If the handle of this knife is squared at the end so as to have two edges, like the end of a microscope slide, it can be used for scraping periosteum and fascia from bone and thereby displaying the attachment of tendons and ligaments. The other knife is for more delicate work. The end of its handle should be smooth and rounded, so that it can be used as a separator. If necessary, a few turns of adhesive tape can be wound around the handle to afford a friction surface. The blade of the knife should be narrow throughout. Some convexity is necessary because a straight edge tends to hook up the tissues. The convexity should, however, be slight and the point well tapered, rather than well rounded. A good blade is made thin, except at the back, by being hollow ground. This enables it to be sharpened readily. Its rigidity depends upon the thickness of its back. In dissecting, the end 5 mm. of the blade are used almost exclusively, and while in use, they should retain the sharpness of a razor. For this, a few strokes on the sharpening stone must be made many times a day. Make incisions with the belly of the blade; dissect with the end 5 mm.; sever with the heel any tough tissues that would blunt the cutting edges.

The Sharpening Stone.—Every possessor of a knife should also be the possessor of a sharpening stone. The stone should be flat, hard and of fine texture (carborundum is satisfactory). Soap lather is a suitable lubricant. Those unskilled at sharpening a knife may learn the art by following the moving picture we have made, in which Dr. Guyatt demonstrates his method.

Dissection.—In dissecting, the rule is to begin by locating the important structures and those in danger of damage. Nerves are, for the most part, more important than vessels; motor nerves, than

sensory nerves; arteries, than veins. Where there are so many veins that "the wood cannot be seen for the trees," let all but the chief ones go, thereby saving time and obtaining a clearer picture, e.g., in the axilla, keep only the axillary and cephalic veins. If an important artery has been destroyed, save its companion vein as a substitute. Cutaneous nerves commonly pierce the deep fascia accompanied by arteries, which, if well injected, serve as guides to the less conspicuous nerves. Every dissection should, so to speak, be set in a frame composed of prominent local structures—such as, the borders of muscles, main arteries, and bone—from which one can get one's bearings. After the contents of the frames have been identified, the areolar tissue and fat should be removed from the muscles and the vessels and nerves should be cleaned. A clean dissection in its frame impresses the eye. The dissection should be completed by defining the bony attachments of the tendons. Doing so at the end of a dissection takes no time; it immensely improves one's knowledge of the bones; and it reveals the direction of the pull of the muscles and perhaps their actions. Ligaments should be defined similarly.

It is waste of time to clean unidentified structures.

Where two are working on the same part, one should spend some of his time in reading, in examing other specimens, in the museum, or in preparing himself for his turn at dissection. The thoughts behind the knife count more than the mechanical process of dissecting.

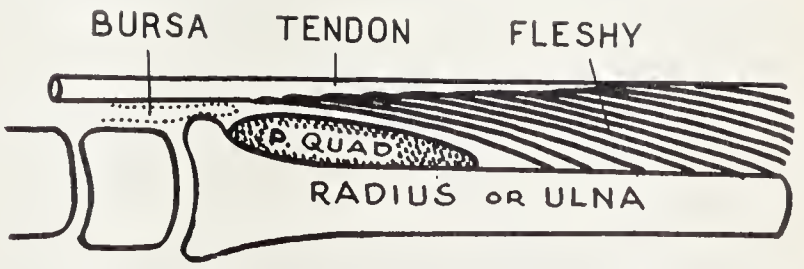
As a routine, the last 5-15 minutes of each dissection period should be spent in naming and handling structures, in restoring relations that have been disturbed, and in reviewing the work of the day.

A knowledge of Anatomy is not dependent upon a good memory. When a student finds that he is endeavouring to memorize his work, he had better take a day off and then start afresh on a different line of study. His memory will fail him; it can hold but a certain amount, but his capacity to appreciate, to correlate, and to apply principles is unlimited. He should endeavour to see things in perspective. He must be able to relate the deeper structures to the surface of the body. He should cover the prescribed field of work systematically. In time he should learn to forget, and having forgotten, to be able to recall. Learning names is not learning Anatomy. A few examples taken from the upper limb may illustrate my meaning.

Perspective.—The junior student is aware that a digital artery and nerve run together on the sides of each finger. He probably knows that (1) the nerve is in front of the artery, and that (2) both lie on the side of the fibrous flexor sheath immediately in front of the margins of the phalanges—and the margins are palpable. A longtitudinal incision, therefore, that strikes bone, leaves the artery and nerve intact, whereas one that strikes the sheath has good prospects of severing them. Obviously it is useless to know fact 1 without knowing fact 2. Facts should be considered in accordance with the magnitude of their importance.

Principles or Generalizations.—The fleshy fibres of an ordinary muscle are of approximately the same length. The fleshy fibres of muscles are too highly specialized to withstand pressure such as a bone or ligament might exert, and when subjected to pressure, they give place to tendon. Tendons cannot withstand friction without becoming frayed, so where they play across a bony surface, ligament or other tendon, a lubricating device in the form of a bursa is invariably interposed. These laws are as unalterable as, say, the laws of optics. (Figure below).

Now, the Fl. Digitorum Profundus and Fl. Pollicis Longus arise from the front of the ulna and radius and their fibres are of equal length, therefore, their tendons must begin on their anterior sur-

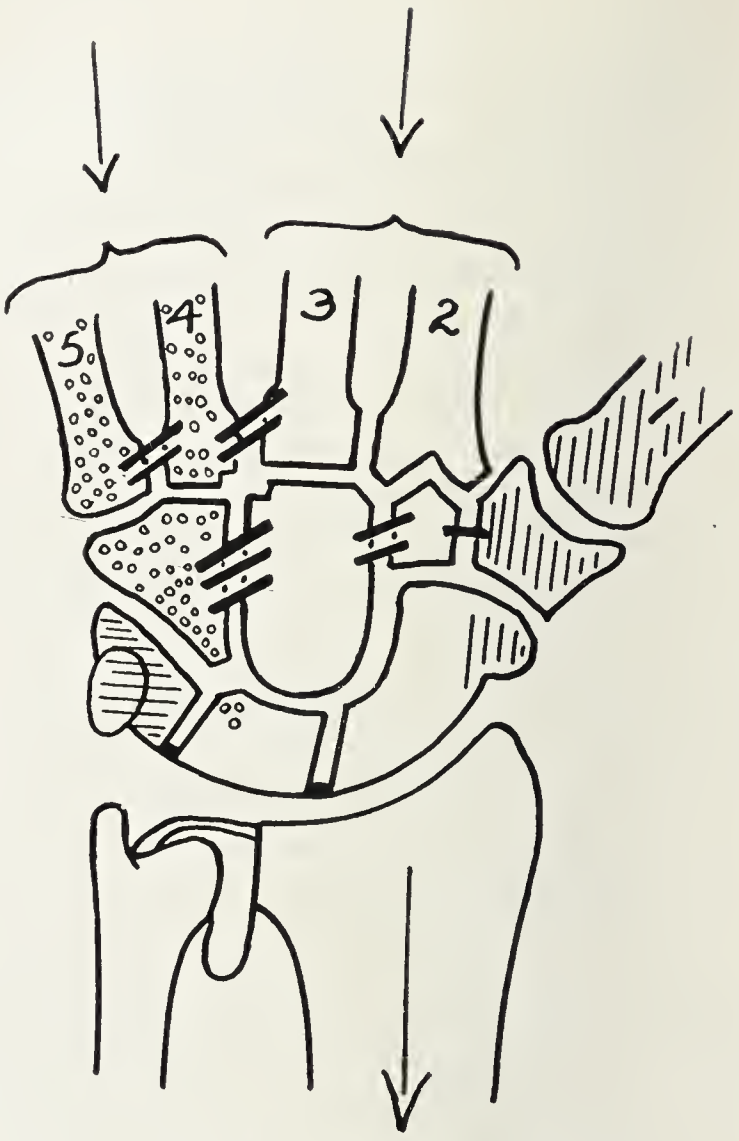


FLEXOR PROFUNDUS DIGITORUM

faces. Their posterior surfaces play across the anterior margin of the lower articular surface of the radius, which is rough, crested, and easily palpated, therefore, the fleshy fibres must cease at this level and give place to tendon and a bursa (the upper end of the palmar bursa) must be present at this site. These facts follow each other in sequence and they could be anticipated.

Common Sense.—Our books tell us to search for certain cutaneous nerves at prescribed (horizontal) levels. But these nerves outcrop on the surface between longitudinally running tendons and muscles, so common sense tells us to look along certain vertical lines. Common sense warns us not to try to study bones without regard to soft parts, because the bones are fashioned by the soft parts and, therefore, cannot be studied intelligently independently of them. Common sense tells us that between flat muscles and underlying parts there must be much loose areolar tissue to allow of freedom of movement. And from a knowledge of the action its amount can be anticipaed during a dissection.

Appreciation and Correlation.—You may become more friendly with the carpal bones if you consider the mechanism involved in striking a blow. When you strike a blow, you instinctively employ the knuckles of the 2nd and 3rd metacarpals in preference to those of the 4th and 5th. Why is this? Because (a) the 2nd and 3rd are long, stout and strong; whereas the 4th and 5th are shorter and more slender. (b) The bases of the 2nd and 3rd are individually equal in surface area to the combined bases of the 4th and 5th. (c) The 2nd and 3rd are rigid and immobile, whereas the 4th and 5th are mobile. Of this you can satisfy yourself by grasping the knuckles and moving them backwards and forwards. (Next figure). (d). The line of force travelling along the 2nd and 3rd is transmitted directly to the radius via trapezoid and scaphoid and via capitate and lunate. The pisiform and triquetrum are not in the line of force transmission. Forces travelling along the 4th and 5th are transmitted via the apex of the hamate to a linear facet on the lunate and so to the radius. (e) This, surely, is a precarious contact. Muscles can render no aid. Security must, therefore, depend on ligaments, because it is on these three—bone, muscle and ligament—that joints rely. Powerful ligaments are, therefore, to be expected, passing from metacarpals 5 and 4 and the hamate to metacarpal 3 and the capitate. Their fibres do not take the transverse direction shown in pictures, but the direction of usefulness, i.e., they are so set as to withstand a blow or thrust.



WHEN STRIKING A BLOW
USE METACARPALS 2 AND 3

Here, then, a dozen facts are linked together. Doubtlessly, they will be forgotten, but they can readily be recalled.

In conclusion, then, dissection is but a means to an end. That end is not achieved by a futile effort at remembering a mass of unrelated facts, but by the acquisition of a habit of mind that concerns itself with the understanding of Anatomy. It has been my intention, briefly, to point out how this understanding renders the study of Anatomy a pleasure, rather than a weariness of spirit.

VARICOSE VEINS AND LYMPH STASIS

By JOHN HAROLD COUCH, M.B.,
Department of Surgery, University of Toronto.

Many patients with huge varicose veins have no varicose ulcer, while many patients with large, stubborn ulcers have no varicose veins. Therefore, in most cases of ulcer some other factors must be in part responsible.

In the study of a large number of ulcers at the clinic for the treatment of veins and ulcers one cannot but be impressed by this observation. Furthermore, many cases of "bad legs" have a few small varicose veins but it is obvious that even if these few were successfully cured the patient would still have definitely "bad legs."

Clinical Picture—Small Fibrosed Legs.

So-called "bad legs" seem to fall into two groups which for descriptive purposes may be called "small bad legs" and "large bad legs."

The small legs are fewer in number than the large ones. They are hard, and covered by shiny skin which may be deeply pigmented and brown, or may be pink and thin. This thin, hard, scar-like skin is tightly adherent over the tibia. The lower half of leg is, throughout its whole circumference, hard and unyielding like a wooden bannister post, while the foot below is relatively normal. The leg immediately below the knee is also nearly normal and the thigh is usually not remarkable.

Ulcers, if present, are hard as to edges and base, and are densely adherent, so that they cannot be shifted. Often covered by an indolent, thin slough, they are surrounded by the above-mentioned wide area of hard fibrosed tissue.

It goes without saying, that these ulcers are difficult to heal.

The pathological process responsible for their appearance must be understood before any logical plan of attack can be launched against the ulcer with hope of lasting success. Even after it is healed, the leg is still small, hard, wooden, far from perfect.

Stasis Factor in Production of Fibrosed Legs.

As a result of the combined action of several factors to be mentioned below, a stasis is brought about in the circulation of lymph from the leg. Although it is true that blood is responsible for the transport of nutriment to all parts of the body and the ultimate removal of all waste products of cell metabolism, it is equally true that the actual tissue cells of the body are not bathed by blood, but by lymph moving slowly in tissue spaces. Any failure of the lymph to circulate, even though slowly, means an impairment in the vitality and resistance to infection of the local fixed tissue cells.

Furthermore, lymph is itself a good culture medium for fibrous connective tissue as has been shown in tissue culture experiments. The tissues in the leg are, first of all, only oedematous. The local connective tissue finding itself in a good culture medium grows, out of proportion, and the swelling which was once only lymph is now lymph plus fibrous connective tissue. The oedema, which once pitted readily and disappeared on elevation, now pits only slightly and is not completely dispersed by elevation.

The over-growth of fibrous tissue continues and the fibrous tissue thus formed contracts as is its wont, so that the leg is infiltrated with a hard, dense white connective tissue which squeezes out nearly all the lymph and blood from the area. The lymph thus squeezed off by fibrotic process finds its way into relatively normal tissues more proximal, or deep to deep fascia and thence upward into the body. The ready clearing of lymph from the leg hypothecates,

obviously, complete patency and adequate functioning of all lymph channels above the fibrosed zone. The higher lymph channels are relatively unharmed. Thus is produced the small hard fibrosed leg.

Clinical Picture—Large Stasis Legs.

The clinical picture of the "large bad legs" is similar in some respects to that of the small specimens. These legs are huge, round, hard, solid posts, like the wooden post at the bottom of the bannister. The skin is thick, oedematous, orange skin, hard and yet showing some slight degree of pitting on pressure. They, too, may be deeply and permanently pigmented. The whole tissues of the leg are resistant, indolent, surrounded by an indurated area extending up to the knee, or even higher.

Ulcers, if present, are large and deep, walled around by great swollen edges; the discharge is profuse and dirty. Granulation tissue is present, but swollen.

Elevation or elastic bandages will result in slow improvement as to size so that ulcers are more shallow, indicating that part of the size is due to oedema.

Stasis Factor in Lymphoedematous Legs.

The element of lymph stasis is large in the production of huge post-like legs, just as it is in the fibrotic type and it is brought about by the same general causes. Here, too, the overgrowth of fibrous tissue goes on and changes oedema to "fibrous oedema." The whole leg remains large and boggy, however, because the return of lymph from the leg as a whole is not free. There is a widespread lymph stasis. The return is not free, because lymphatics are damaged higher up in the leg.

As an illustration of this process, one might call to mind the oedema and elephantiasis occurring in filariasis or when pelvic lymphatics are harmed by the spread of carcinoma from a uterus, or by the fibrosis in the pelvis consequent upon heavy radiotherapy for such carcinoma.

The stubborn and even permanent lymphoedema persisting after phlegmasia alba dolens is also an evidence of destruction of lymphatics in the pelvis, which accompanies thrombosis (perhaps infected) of pelvic veins nearby and which is at times an unfortunate complication of confinement or operation. Most are now agreed that thrombosis of vein alone will not produce the clinical picture of "milk leg."

The fundamental process behind both sorts of legs is then, the same, namely, lymph stasis and its consequences. Essential differences between the two, however, do exist. In the small legs, fibrosis predominates and lymph return from the leg, as a whole, is possible. In the large ones, fibrosis and oedema are both present and lymph return from the leg is imperfect. We have seen a large oedematous, hard, painful leg converted into a small, hard, but considerably more satisfactory leg, by the removal of deep fascia, together with diseased superficial fascia. Herein lies the clue to the differences between the two as well as the clue to treatment.

Lymph Stasis.

Having recognized the differences as well as the similarities in the types, we may study the processes which precipitate or encourage lymph stasis itself.

The mere presence of varicose veins, with their slow downward flow, tends to permit stasis of fluid in tissue spaces, although the facility and reserve of the return mechanism is amazing, so that it continues to function efficiently in the face of difficulty for a long time.

Phlegmasia alba dolens is a specific type of damage to the lymphatics, which are so closely associated with the large pelvic veins. That the clinical picture is in a large measure due to lymph stasis, rather than venous obstruction, is suggested by the appearance of the leg. This is large, pale, heavy, and pits on pressure, instead of being blue and cold, with dilated collaterals, as might be expected if purely venous. Experimental work is reported which further

confirms this suggestion. A fair number of our patients with stasis ulcers give a history of milk leg.

Cellulitis of a low grade is a repeated incident in legs, the skin and cellular tissue of which is below par. A small scratch or injury provides the portal of entry and infection thus introduced, shows itself as an infected chronic ulcer. More important still, however, is the zone of cellulitis, which surrounds the ulcer and involves the soft cellular tissue of the leg, often over a wide area. These typical subacute attacks of a diffuse cellulitis occur again and again. Each time they are associated with lymphangitis and lymphadenitis, so that repeated infection is a further important factor in producing damage to lymph vessels and lymph nodes, as well as laying down scar tissue locally in the leg and around lymphatics.

Since fibrosis and more or less complete lymph blockage are really two elements in the same process, it follows that there are all degrees and proportions between the two extremes.

Treatment of Lymph Stasis.

Since the essential pathological process behind all the various types of lymph stasis is the same, the principles on which its treatment is based are common to all, and can be discussed together. It is obvious that treatment must be directed, first, toward correcting lymph stasis and, second, towards getting rid of fibrous tissue.

Here, as in patients suffering from varicose veins, the tissues superficial to and even including deep fascia, are the layers chiefly at fault. Under deep fascia, the muscles and areolar tissue appear healthy. All tissue which lies between skin and deep fascia is diseased, and serves no useful purpose. Operation, therefore, removes mechanically, as much of this fibrotic fatty tissue as seems expedient. A strip of deep fascia is also removed, so that the skin flaps are laid back on soft healthy muscle. The deep lymphatics in muscle are unimpaired. Anastomosis takes place as the skin flaps settle into their new bed and the skin is now drained by deep lymphatics. It becomes softer, less oedematous and the leg, as a whole, is smaller, due to the better lymph drainage, as well as to the actual removal of a certain portion of subcutaneous fat.

Not only does this operation result in improved appearance, but what is more important, it results in improved lymphatic circulation from the leg, so that the skin is more healthy and the danger of ulceration rendered less imminent. The patient must realize, however, that although improved, his margin of safety is less than normal, and that constant care on his part is essential if ulcers are to be avoided.

Treatment of Ulcers.

If ulcers are present in a fibrotic or oedematous leg, as is all too often the case, their treatment naturally falls into two parts.

1. *Treatment to Heal.*—An open infected ulcer must always be healed before any operation is performed. The infection is handled by using antiseptic dressings, of which we prefer 25% solution of Hygeol in cold water, because of its ability to dissolve dead tissue as well as its antiseptic qualities. The lymph stasis factor is best handled by putting these patients to bed with the leg elevated. Gradually oedema disappears, circulation improves, infection clears and the ulcer is covered with thin epithelial tissue, eking out a precarious existence on a hard, unfriendly soil.

Many times it is desirable or even necessary that people remain on their feet at work, in spite of ulcers. Fortunately, it is possible to accomplish substantially the same results in a little longer time and still permit patients to be up. The ambulatory treatment is successful, because the same sound principles, which assure success of treatment in bed, are kept in mind and followed, though the means of accomplishment may appear different.

Infection and slough again are cleared by Hygeol dressings. As soon as the ulcer surface is reasonably clean, the

whole foot and leg is bound *tightly* in an elastoplast bandage, the satisfactory application of which is beset by several minor hazards, which are overcome by experience or training, if results are to be happy. The ever-tightening elastic pressure squeezes and milks the oedema out of the leg and at the same time exerts some manner of mild massage in the region of the ulcer. Profuse watery discharge is often squeezed right through the elastic bandage. As the swelling lessens, the elastic bandage contracts and keeps pace with it. After three weeks, however, it should be renewed. Ulcers will heal under this regime.

Treatment to Keep Healed.—When ulcers heal, they are covered by a thin layer of epithelium resting on a harsh bed of scar, which has replaced granulation tissue that once covered the base of the ulcer. The underlying scar contracts and the vessels which reach the surface to nourish epithelium are few and small. The slightest trauma results in another stubborn ulcer.

To excise such a healed area with the underlying scar and to cover it with a Thiersch graft is futile. An environment so uncongenial as to result in death of the original skin will not be found well-suited to the needs of any skin which we may place there, any more than to the needs of the thin scar of a healed ulcer. Before transplanting skin, it is absolutely necessary to make an improvement on Nature — to provide a more nourishing bed than the natural one. For this reason, we excise not only ulcer and underlying scar, but also the deep fascia, so that the new graft now lies on soft healthy muscle and stands a reasonable chance of survival.

The excision and grafting of ulcer is combined with the operation mentioned above for the improvement of lymph return. Thus the new graft is provided with a nourishing bed the surrounding tissues are improved and while the leg is still far from normal, reasonable care on the part of the owner will be rewarded by freedom from pain, swelling, and ulceration for an indefinite period of time.

The excision and grafting operation is one of considerable technical difficulty, and requires meticulous attention to several details if disappointment is to be avoided. It is anticipated that it will be further discussed by one of the members of the clinic who has been particularly interested in this for some time.

FRACTURES OF THE MANDIBLE.

By STUART GORDON, M.S. (Tor.)

My last communication to this journal dealt with fractures of the malar bone. When asked recently to contribute again, it occurred to me that a discussion of the ordinary fractures of the mandible might be acceptable, and that such a study might logically follow the previous paper.

The mandible is the common facial bone to suffer fractures. We are not then dealing with a rare fracture though we are dealing with a fracture which most of us know but casually.

Aetiological Factors.

The aetiological factors of mandibular fractures are those present in any fracture. Generalized disease of bone may be responsible, e.g., fragilitas ossium. A few fractures are the result of local bone disease. Thus a fracture may occur through a dental cyst, an adamantinoma, a secondary carcinoma, or through a sarcoma. The commonest cause by far is trauma, which may act either directly or indirectly. A blow from a fist, not an unusual determinant, may produce its effects at the point of contact, or at a more distant site.

It will be readily realized that such factors as the direction and force of the blow, whether the mouth was open or closed at the time of the injury, and

the number and distribution of the teeth, besides the condition of the bone, will all play a part in determining the site, or sites, of fracture.

Varieties.

A single break, commonly at the angle or through the mental foramen, is the customary fracture. Double fractures are not uncommon. Thus breaks through the mental foramen and the opposite angle are frequently seen. This association is so repeatedly met with, that careful attention should be given to the opposite angle in any fracture through the mental foramen.

A break may occur at any point in the mandible. All, or almost all fractures of the body of the mandible are compound. On the other hand fractures of the ascending rami are never compound, protected as the rami are by muscle.

Diagnosis.

The diagnosis of fracture of the mandible is made on the history, physical examination, and radiological examination. No one of these should be omitted.

The history is usually short and definite. An attempt to crack a nut with the teeth, or some such unusual effort, is followed by sharp localized pain in the mandible which is worse on movement. Or, the patient has received a blow on the lower jaw which has resulted in local pain, swelling, and interference with function.

Usually the diagnosis can be made on inspection alone. If the fracture is in the body of the mandible, mal-alignment of the teeth will be noted. When an attempt is made to open the jaw, it swings to one side or the other. Abnormal mobility is present. One can make sure, by palpation, that the abnormal mobility involves the whole thickness of the bone. Haemorrhage is to be seen in and about the area of fracture.

If the inferior dental nerve has been interfered with the patient complains of numbness of lip and gum, distal to the injury.

X-rays should be taken of the whole mandible, not only of the area of suspected fracture. The exact extent of the injury is determined thereby and adequate treatment made possible.

Treatment.

Treatment of a fractured mandible may be very simple, and it usually is, or it may be very difficult. Fundamentally as for all fractures, treatment consists in accurate reduction followed by maintenance of the fragments in good position until union occurs.

Very accurate reduction may be obtained if all the teeth are present since any mal-alignment, i.e., malocclusion of the teeth, is at once appreciated by the patient. It immediately becomes evident, therefore, that to treat mandibular fractures satisfactorily some idea, at least, of normal occlusion is necessary. Of course, in those cases which are treated without anaesthesia, the patient can inform the operator the moment his teeth bite normally.

The upper central incisors are about one-third wider transversely than the lower central incisors. Thus the upper central incisor comes into contact with the lower central incisor and from one-third to one-half of the lower second incisor. Similarly the second upper incisor is in relation to the medial portion of the lower canine. The upper canine is usually broader than the lower. Its lingual triangular ridge fits between the mandibular canine and the outer cusp of the adjacent bicuspid. The outer—or buccal—cusp of the mandibular first bicuspid occludes with the maxillary canine and the first bicuspid. Next the buccal cusp of the lower second bicuspid occludes in the sulcus between the upper two bicuspids. The upper first molar has its medial two-thirds occluding with the outer two-thirds of the lower first molar. The oblique ridge of the upper first molar thus lies between the two lower molars. This arrangement usually holds for the rest of the molars.

The first step in treatment is a thorough cleansing of the mouth. Any

teeth in the line of fracture are removed. Then one decides upon the method of fixation. Cap splints, cemented to the teeth, and capable of being locked, upper to lower, may be used. They are efficient, costly, and require the services of an expert technician. The method in widest use today is the wiring of the mandibular to the maxillary teeth. Silver, stainless steel, or phosphor bronze wire may be used. Ivy's eyelet method is the simplest and is very efficient.

Wiring can be done in most cases without an anaesthetic if the operator is careful and gentle. As already mentioned, the patient can appreciate better than anyone else when his proper bite has been restored. Reduction is thus aided if we can do without the anaesthetic. Right and left bicupids, and the central incisors are the teeth usually used in splinting. Wire is looped about them and tie wires are used to fasten the upper to the lower teeth. If for any reason it becomes necessary to open the patient's mouth, the tie wires are cut, but the loops are not disturbed, and refastening is easy. Wires are left on for a period of five weeks. During this time the patient can only take fluid nourishment. Once they are removed, soft food only is taken during the next two weeks.

Complications.

Complications are relatively rare. Secondary haemorrhage occasionally occurs. It is treated by removing the wires and applying prussure either directly or by packing. Infection is the commonest complication. Its occurrence is favoured by delay in fixation, or by leaving a tooth in the line of fracture. When one remembers that practically all fractures of the body of the mandible are compound, it is surprising that this difficulty is not encountered more frequently. If pus is definitely present, dependent drainage should be instituted.

Non-union is very rare. Delayed union is seen from time to time, and, as usual, is the result of lack of approximation or of fixation.

Conclusion.

Fractures of the mandible are not uncommon. In the main, treatment is simple. No attempt has been made in this paper to deal with unusual fractures. Simple, fractures properly fixed and maintained in good position, yield uniformly good results.

A **NEW** WORK BASED ON **30,000** CASES

SCHUMANN'S

TEXTBOOK of OBSTETRICS

Dr. Schumann's book is thoroughly up-to-date; you will not be disappointed upon any phase of new and accepted practice on which you may seek information. This work is accurate, modern, authoritative and, above all, practical and clinical, and gives *EVERY STEP OF OBSTETRIC MANAGEMENT.*

Textbook of Obstetrics. By Edward A. Schumann, Professor of Obstetrics, University of Pennsylvania. 780 pages; 581 illustrations on 497 figures. $7.00.

McAinsh & Co. Limited

Dealers in Good Books Since 1885

388 YONGE ST. - - **TORONTO**

APHASIA

By H. H. HYLAND, M.D., M.R.C.P., F.R.C.P. (C.)
Department of Medicine, University of Toronto, and the Medical Service, Toronto General Hospital.

Aphasia can be defined as a disorder in the comprehension and expression of meanings by the use of symbols employed in speech. The disorder results from a lesion in the region of the cerebral hemispheres which has to do with speech. This definition clearly distinguishes aphasia from dysarthria, the latter being a disturbance of articulation resulting from weakness or inco-ordination of the muscles concerned in the production of vocal sounds. Thus dysarthria occurs in diseases specifically affecting the muscles, e.g., muscular dystrophies; in diseases of the peripheral nerves, e.g., polyneuritis; in diseases involving the cerebellum and therefore affecting the co-ordination of the muscles, e.g., multiple sclerosis; in diseases affecting the cranial nerve nuclei in the brain stem, e.g., progressive bulgar palsy; in diseases of the corpus striatum, e.g., paralysis agitans; and in diseases associated with bilateral involvement of the upper motor neurone, e.g., dementia paralytica or bilateral hemiplegia. When dysarthria is present, the words may be slurred and indistinct but the patient does not use wrong words or have difficulty in comprehension such as occurs with aphasia.

It is helpful in the understanding of aphasia to consider the sequence of events during the development of speech. The infant first learns to communicate with the outside world by means of gestures; later, he learns to associate certain types of sound with the objects he perceives by means of visual, tactile, auditory, olfactory and gustatory senses. By endeavouring to imitate these sounds, associations are developed between kinaesthetic sensations from the muscles of articulation and the sound of the speaker's voice. Adjectives, verbs and prepositions are learned later than nouns, and their learning requires an association of sounds with a certain degree of abstract thought beyond the simple perception of an object. Reading requires the association of visual signs with the sounds which have been learned. In writing, movements of the hands are employed to reproduce visual signs similar to those forming the basis of reading. The whole represents the complex mode of expression known as language. It must be emphasized that speech is not a faculty. It consists of many elements, including well coordinated memories and symbols. As stated by Pierre Marie: "It is the means whereby intelligence finds its readiest expression but it does not constitute the whole intelligence."

In aphasia, the disturbance is in the comprehension, elaboration and expression of speech symbols, not simply a dissociation into faculties of reading, writing and speaking. In every case, the whole domain is affected although usually one or other of comprehension, elaboration or expression is most involved.

In 90 per cent of individuals, the predominant rôle in speech is played by the left cerebral hemisphere. The region of the hemisphere which is most particularly concerned in speech is the external convexity of the temporal lobe and an area extending upwards to include a portion of the parietal lobe above the Sylvian fissure, spreading backwards in the supramarginal and angular gyri and forwards over all the convolutions of the insula to the posterior ends of the second and third prefrontal gyri. This speech region of the brain comprises not only the cortex but also the subcortical white mat-

ter. By means of association fibres underlying the cortical grey matter, the various groups of cortical cells are brought into intimate association with each other. Thus, the visual association area in the occipital lobe, the auditory association area in the temporal lobe, and the cells in the postcentral gyrus receiving kinaesthetic impulses from the muscles of articulation and the upper limb are all connected with each other and with the efferent pathways for speech. For example, the fibres in the external capsule are important in linking the posterior half of the hemisphere to the lowest part of the precentral convolution. By this means the comprehension of speech symbols is connected with cells having to do with exteriorization of speech, and interruption of this pathway causes severe dissociation of function.

Aphasia can be broadly classified as congenital or acquired. Congenital word deafness is a rare disorder but very important to recognize. These children hear perfectly well, but are unable to appreciate the significance of what they hear. It is supposed that the anatomical mechanism is lacking whereby sounds become associated with other sensory impressions and so acquire meanings. It is thought to be due to an aplasia of the posterior temporal lobe cortex on both sides. For years the child may not speak at all, but eventually it acquires a vocabulary of its own which is incomprehensible to strangers (idioglossia). These children are found usually in institutions for mental defectives although they do not necessarily suffer any defect in mental capacity.

Congenital dyslexia or defect in learning to read is of two types: (1) lack of ability to appreciate visual symbols; (2) visual symbolization is intact but there is mirror reading and writing associated with left-handedness. There is usually no general defect in intelligence.

Children suffering from congenital word deafness and congenital dyslexia can usually be trained by reeducation to overcome their disabilities and for this reason it is very important that they be not regarded erroneously as mental defectives.

Acquired aphasia is usually the result of vascular, neoplastic or inflammatory lesions of the brain. In the great majority of individuals, either the right or left hemisphere plays the dominant rôle in the integration and operation of the language mechanism. The hemisphere is determined by being contralateral to the preferred hand and in 90 per cent. of people it is accordingly the left hemisphere. However, there are certain exceptions to this rule which merit some consideration.

Chesher has reported a study of 164 cases of aphasia. Of these, 157 were completely right- or left-handed and the lesions were verified as being in the hemisphere opposite to the preferred hand. In these latter cases, therefore, the ability to formulate and express language seemed dependent on a factor operative in only one cerebral hemisphere. The remaining 9 cases gave a history indicating mixed motor preferences: e.g., in each case one hand was preferred for certain acts but the other hand also was preferred for other equally important acts. These 9 aphasic patients were found to have lesions in the right hemisphere in 5 cases and in the left hemisphere in 4 cases. It was concluded that in these 9 cases the language mechanism was unlateralized and that a lesion in either hemisphere would have resulted in aphasia.

The importance of realizing that the speech region may be unlateralized in certain individuals is well illustrated by a case recently seen in the Toronto General Hospital, where difficulties in localization of the lesion were encountered.

Case D.D., aged 26, admitted to hospital March 20, 1936, complaining of discharging ear for five days, headache for three days, difficulty in speaking for two days. Three weeks before admission, patient was confined to bed for a week with a severe cold. On recovery, he returned to work until five days ago, when he developed severe pain in the left ear, and eighteen hours later, the ear com-

menced to discharge. Examination showed spontaneous speech to be unintelligible, comprehension of the spoken voice was poor, so that he was unable to carry out simple commands. He could not understand written speech and was unable to write spontaneously or on dictation. There was slight stiffness of the neck; a right facial weakness was detected on voluntary movement, and sensation was mildly impaired over the upper part of the right face. Allowing for the poor co-operation of the patient, it was thought that a defect was present in the right temporal visual field.

The interpretation of the finding of aphasia in this case was difficult, because the patient was completely left-handed and it was the left ear that was involved. No family history of left-handedness was obtained, but it was interesting that his five married brothers and sisters, all right handed, have an average of four children in each family and in every one of these five families there is one child that is left-handed.

Because of the presence of the right field defect and the right-sided facial weakness, it was decided that the patient had a lesion in the left temporal lobe, but not a frank abscess. A left-sided mastoidectomy was carried out, but during the next few days, his condition became worse. The weakness of the right face increased and the right arm became very weak. He had several convulsions with deviation of the head and eyes to the right.

On March 26, gradual improvement commenced, so that by April 1, he could comprehend what was said to him, although spontaneous speech was still poor. The aphasia steadily improved, however, and by April 13, it was no longer obvious and there was no weakness of the arm or face and no field defect. He was discharged from hospital on May 3.

Patient was re-admitted on June 7, because of a recurrence of headache during the past week and the sudden onset of drowsiness on the day of admission, leading to unconsciousness within a few hours. Death occurred on June 9, with all the clinical evidences of a severe purulent meningitis.

Autopsy showed a frank purulent meningitis, but, in addition, there was a moderately sized area of softening in the infero-lateral surface of the left temporal lobe. Microscopically, this area showed a partial degeneration of the cortex and white matter with much cellular infiltration. It was considered probable that this localized area of encephalitis was the likely source of the terminal meningitis and the original cause of the cerebral symptoms.

Obviously the speech mechanism was not strictly lateralized to the right hemisphere in this left-handed patient, and it is important to remember that this sometimes happens in left-handed patients, where no family history is obtained of left-handedness in preceding generations. The rapid recovery from the aphasia may have been due, in part, to the subsidence of oedema in the vicinity of the encephalitic process in the left temporal lobe, but as the encephalitis remained active, the complete nature of the recovery can best be explained by a predominance of the speech mechanism in the right hemisphere associated with left-handedness. Had the lesion occurred in the speech area of the right hemisphere in this patient, it is probable that aphasia would have been present and that recovery would have been less complete than actually was the case.

Until recent years, it was thought that much localizing value could be placed on the type of aphasia in a given case. The long continued belief in Broca's area in the left inferior frontal convolution as the centre for motor speech is an example of this strict localization. No doubt, lesions in this situation are sometimes associated with motor aphasia, but it can be readily ascertained that most specimens show widespread lesions involving much more of the brain than this alleged motor speech area. According to Burkhardt, Broca's area has been extirpated in man with no aphasia resulting. Pierre Marie concluded from extensive pathological studies that aphasia and lesions of Broca's area were rarely found together, and, when they were associated, the lesion usually extended subcortically.

Pure word blindness and pure word deafness, as acquired conditions are unusual, but do occur occasionally as a result of small lesions undercutting the subcortical fibres from the left parieto-occipital region and the posterior part of the third left temporal convolution respectively. It is obvious that such discreet lesions confined to limited areas of the subcortical white matter are rare and it is important to realize that these conditions, occurring as isolated phenomena, could only result from a subcortical lesion and never from a lesion involving the cortex.

Head has shown that any cortical

lesion of sufficient size to cause aphasia depresses the functions of speech as a whole. The same type of disturbance underlies both comprehension and expression in aphasia and consequently any narrow localization is impossible. In general, it is true that lesions situated behind the Rolandic fissure disturb comprehension of speech chiefly, whereas lesions in front of the Rolandic fissure disturb mainly the expression of speech, but localization, based on the type of aphasia alone, should not be carried further than this.

Considerable defect in general intelligence always accompanies severe damage to the speech area because of the large rôle played by speech patterns in the workings of the human mind. But even with the most severe degree of aphasia patients are able to communicate with others by means of gestures, as this primitive form of conveyance of meaning is always retained.

Head's classification of aphasia represents a great advance in the conception of the condition and is based on the order of appearance of speech defects in the presence of increasingly extensive lesions in the cortex and subcortical association pathways. Space will not permit any detailed consideration of this classification but, briefly, he recognizes four main types of aphasia.

Verbal aphasia is the commonest type encountered. The expressive aspect of speech is chiefly affected, propositional being much more involved than emotional. Reading and writing are poorly performed and there is difficulty in grasping complex meanings.

Nominal aphasia. The patient is unable to name objects but the structure of words is correct. There is difficulty in comprehending written and spoken speech. This type of aphasia is commonly not recognized for what it is, being sometimes mistaken for a general intellectual impairment which, superficially, it may resemble.

Syntactical aphasia is characterized by a grammatical incoherence and in this way can be distinguished from verbal aphasia in which grammar is correct. The patient talks a voluble jargon because he fails to understand what he himself is saying.

Semantic aphasia. Articulated speech is not mutilated but the patient fails to understand what is said to him or what he reads. This speech defect merges into a defect of general intelligence.

Verbal and nominal aphasia are usually associated with lesions anterior to the Rolandic fissure, whereas syntatical and semantic aphasia are associated with lesions posterior to the Rolandic fissure.

The prognosis in aphesia is good when it is the result of lesions pressing on the hemisphere from outside and if this pressure can be relieved, e.g., depressed fracture of the skull, menigioma, subdural haematoma. It is poor with lesions which destroy the cortex and subcortical white matter, e.g., extensive vascular lesions and infiltrating tumours. Inflammatory lesions of the non-purulent variety do not often cause aphasia but when they do the prognosis is usually good if the patient survives, probably because much of the disturbed function of the brain is due to oedema. Purulent inflammatory lesions, such as abscess, have a good prognosis if removal can be carried out easily. Hemiplegia due to a vascular lesion in the left hemisphere is usually associated with a marked disturbance of speech at the onset. If the patient survives, the disturbance frequently improves markedly and occasionally recovers completely. When recovery takes place, it can be assumed that the cortex is not involved in the lesion and that the speech disorder is the result of damage to association fibres, possibly in the external capsule, or those from the lower part of the left frontal lobe passing to the opposite side via the corpus callosum. Involvement of the latter fibres disturbs the bilateral coördinating mechanism of the muscles of articulation and the resulting speech disturbance will have features suggesting dysarthria rather than aphasia.

Lesions involving association fibres

do not usually produce permanent severe symptoms unless they are very extensive, seemingly because undestroyed fibres can take over additional function if the occasion arises. Lesions causing severe cortical damage, however, do not show the same recuperative capacity and the prognosis is worse in proportion to the severity of the aphasia, its duration, and the age of the patient. It can also be stated that in general the prognosis is worse where the receptive type of aphasia is present than in cases where expression is most affected.

REFERENCES.

Brain, Russell: Textbook of Neurology, London, 1933.

Oorton, S, T.: Word blindness in school children. Arch. Neur. & Psychiat., 1925, xiv, 551.

Cresher, E. C.: Bulletin Neur. Inst., N.Y., 1936, iv, No. 4.

Head, Sir Henry: Aphasia and kindred disorders of speech. Brain, 1920, xliii, 87.

Aphasia: an historical review. Ibid, p. 390.

Aphasia and Kindred Disorders of Speech 2 vols.), Cambridge, 1926.

Wilson, S. A. K.: Aphasia, London, 1926.

DISSEMINATED SCLEROSIS

By I. J. TEPPERMAN, B.A., '37.

Disseminated (multiple or insular) sclerosis is one of the commoner neurological diseases, yet its etiology is still obscure. In 1837 there appeared Carswell's sketch-book of pathological specimens seen in a London hospital, one of which was a spinal cord with the pons and medulla showing superficial coloured plaques seemingly placed indiscriminately throughout the parts. This is described as a "peculiar diseased state of the cord and pons, occupying the medullary substance, which was hard, semitransparent and atrophied, beginning on the surface of the white and extending to the gray substance." In the same year Cruveilhier described a 'sclérose on îles' appearing at the expense of the normal white cord-tissue which it replaced at times almost completely. "This new tissue is dense—much more so than the cord itself. I cannot compare this tissue with any other morbid tissue of which I have knowledge." He called the clinical condition "induration of the cord with paraplegia." In the 60's and 70's Charcot and his school crystallized the earlier clinical and pathological observation and published the symptomatology and pathology much as we have it to-day. Since that time the advance has been largely on the clinical side with the addition of minutiae and the nicer points of diagnosis. The last fifteen years has shown a marked upsurge of interest in the etiology and histopathology of the disease, with tangible results.

Incidence.

Disseminated sclerosis makes up 2-3% of organic nervous disease, and appears to be, after syphilis, the most frequent disease of the nervous system.

Age: about 95% of the cases occur between the ages of 14 and 40, and 75% between the ages of 20 and 35. It is very rare in children, though a few cases are reported, some as young as 3 and 4. There are exceedingly few if any authentic cases of disseminated sclerosis with onset after 45.

Race: no racial tendency can be described though the negro appears to be less subject than the white.

Nationality: the disease is apparently rare outside Europe and North America. It appears to be most common in Switzerland, where a survey showed it to be present in 1 in 4,000 population. In England and Wales it is found 1 in 8,600. In the United States it is about one-third as common as in Great Bri-

tain. It is common in Norway, rare in Italy and Roumania, very rare in South America and South Africa, and appears to be unknown in the natives of India, China and Japan. The degree to which the population of the tropics is spared is striking.

Family: beyond the occasional occurrence of the disease in two members of the same family, there is nothing to suggest an hereditary or familial origin.

Sex: according to the literature the male is more often affected—in the ratio 3:2. However, it seems more likely that male and female are equally likely to develop the disease.

Occupations there is no satisfactory statistical evidence of any occupational incidence of the disease.

Environments there appears to be a preponderance in urban rather than in rural dwellers.

Etiology.

The essential lesion in disseminated sclerosis is a primary patch of demyelination of the brain and cord followed almost simultaneously by a secondary gliosis or sclerosis resulting in a varying degree of destruction of the nerve-cells and their axis-cylinders. Two main theories of the origin of the disease have been suggested: A. *Theory of Endogenous Origin*, which states that disseminated sclerosis is the result of some developmental or congenital defect of the neuroglia, which is thus rendered more liable to be affected by irritation than is normally the case, i.e., gliosis is primary and the glial overgrowth compresses and destroys the myelin sheath. This theory has never found many supporters since demyelination is known to procede gliosis.

B. *Theory of Exogenous Origin.* Various external causes have been suggested as the etiological factors in disseminated sclerosis.

(1) *Infections* since Maria (1891), various neurologists have entertained the opinion that disseminated sclerosis may be the sequel of the most common acute specific infectious diseases, such as measles, scarlet fever, typhoid diphtheria and others. It has been observed in certain cases that the symptoms of disseminated sclerosis appeared soon after the occurrence of an infectious disease in the same person, and there is no doubt that an existing disseminated sclerosis may undergo exacerbation from intercurrent infection. There is nothing to support the idea that focal infection plays a part. Syphilis and tuberculosis positively play no part in the etiology. The spirochaetal theory presented especially by Steiner shows at present the balance of evidence on the negative side. Noguchi (1923), using his vast experience in spirochaetal research, inoculated large numbers of rabbits and guinea-pigs in every conceivable way with completely negative results; while repeated dark-field examination of the material revealed no spirochaetes. It must be remembered that spirochaetes of various kinds are normally present in most animals used for experimental purposes. The virus Sphaerula insularis of Chevassut (1930) has been completely discredited. But many English neurologists are of the opinion that disseminated sclerosis is probably due to a virus belonging to the same group as those causing post-vaccination and post-exanthematous encephalitis but that the evidence does not prove that it is the same virus.

(2) *Chemical Poisons*: Oppenheim suggested that mineral substances, especially lead, arsenic and tin, might be responsible for disseminated sclerosis. Cone (1934) revived the idea, being able to show lead by X-ray in the skeleton of some cases of disseminated sclerosis. However, there is very little to bear out his contention. In a great number of cases the disease begins early in life, often at a period before the patient can have been subjected to occupational poisons.

(3) *Trauma*: patients who suffer from chronic nervous disease are prone to incriminate trauma, physical or mental, as the causative agent. It must be remembered that injuries often occur in disseminated sclerosis as a result of, rather than the cause of, the disease. There is no doubt, however, that dissem-

inated sclerosis may suddenly become manifest after severe physical or psychic trauma; and that existing disseminated sclerosis may undergo marked exacerbation after trauma. Allison (1931), in his survey of North Wales, denies any relation between injury and the onset of symptoms.

Russell Brian in his critical review of disseminated sclerosis states that all the evidence points to disseminated sclerosis being an infective disease. However, in the five years since Brain's statement in 1931, a great deal of experimental work has been done on experimental demyelination and its possible etiological relation to disseminated sclerosis.

Putnam (1936) sums up all his experimental work in a very recent article: he admits that the various experimental ways of producing plaques of demyelination prove little in regard to the human disease, but do suggest that (a) a local venous obstruction should receive consideration as the immediate cause of the primary demyelination, (2) the process may be precipitated by an organic toxin and probably by any one of many, (3) some fundamental disorder of the blood leading it to clot in the venules of the central nervous system as the result of stimuli such as infection, pregnancy or emotional upsets which normally would lead only to a mild increase of the coagulability of the blood. Exacerbations of disseminated sclerosis are almost constantly produced by pregnancy, in which the coagulability of the blood is increased.

From it he postulates a myelinolytic factor in the blood of patients with disseminated sclerosis. However, Weil and Cleveland (1932) showed that other diseased plasma besides that of disseminated sclerosis could cause myelinolysis in rats' cords and concluded that the increase of lipase in the blood of many patients with disseminated sclerosis was of little value in the etiology.

Morbid Anatomy.

The following is a picture compounded from various pathological studies, and perhaps is the simplest. The pathological "unit" in disseminated sclerosis is a circumscribed patch of nervous tissue in which the pathological process runs a fairly well-defined course, terminating in a sclerotic plaque.

Early Patch: the majority of observers agree that the patch is in many cases placed concentrically around a vessel. Plaques may be of microscopic size, about a small dilated venule, and extending along it like a sleeve; or they may be several cms. in diameter. The changes seen from the center of the early patch to the periphery are as follows: The blood-vessels are dilated, but their walls are unchanged. In the perivascular spaces of Virchow-Robin there is a dense accumulation of cells of several different types—a few lymphocytes and plasma cells, but the great majority are the very numerous fat-granule (also called scavenger, gitter, compound granular corpuscle cells) cells, in reality modified phagocytic microglia cells, which on staining with osmic acid are seen to contain in their cytoplasm globules of degenerating myelin and axonal debris. Around this perivascular space is a concentric zone in which the myelin sheaths of the nerve fibres are severely damaged—some have disappeared, others are swollen and show large or small discrete fat droplets. Filling up the spaces between the swollen parallel axones are proliferating astrocytes with numerous processes, and a few lymphocytes and fat-granule cells. The axones persist for the most part either unscathed or showing degenerative changes such as swelling, twisting, fragmentation, inequality in size. Peripheral to this zone there is a gradual transition to the normal tissue with less disintegration. As a rule the patches are confined to the glia-bearing portions of the nervous system. The spinal nerve roots are frequently involved in the gliosis.

Late Patch: this is merely the end result of the changes in the early patch. The fat-granule cells have disappeared, probably having discharged their contends into the capillaries, and leaving the perivascular spaces dilated. The

blood-vessels may appear perfectly normal or show some hyaline thickening and some proliferation of the adventitia. Most of the axones show complete demyelination, many are partially demyelinated, and the spaces between the axones are filled with a condensed glial network. These axones are also reduced in number and many of the remaining ones show some abnormality, such as varicosities, fusiform swellings, atrophy. In the transitional zone there is a narrow ring of large glial cells. Meningeal changes are variable, the pia usually showing a slight thickening and lymphocytic infialtration. Old plaques may show a lighting-up of the process paripherally so that all the various stages of the disease may be found in the same plaque. The smaller the plaque the more sharply it is differentiated from its surroundings. In the cord the ventral horn cells are not involved in the gliosis. In short, the three consecutive stages of patch evolution are, swelling and fragmentation of the myelin, a stage of microglial activity with removal of the myelin, and lastly, the glial scar.

The plaques are scattered throughout both the brain and cord and there may be scores of them. A purely spinal type of lesion is very uncommon. The optic nerve is the cranial nerve most frequently attacked, most often at the chiasma, though the whole intracranial course of both optic nerves may be completely devoid of myelin. Loss of myelin in itself does not prevent the transmission of nervous impulses. A practically complete demyelination of some levels of the brain-stem is compatible with life, and almost complete demyelination of the optic nerves is compatible with vision.

The frequent nuances that exist between the various syndromes in disseminated sclerosis, disseminated encephalitis and Schilder's disease suggest that they are perhaps entities only in the sense that tabes is an entity within neurosyphilis. The probabilities are that these three diseases all belong to the class of non-suppurative infection encephalomyelitis.

Symptomatology.

No exact correlation is possible between the symptoms and lesions in disseminated sclerosis. The extent of the disease as judged from pathological examination is always much greater than the clinical examination would suggest. Indeed one is filled with surprise that all the functions are not abolished.

The commonest early symptom is the development of sudden weakness or clumsiness of one or more limbs, usually the lower. Paraesthesiae, usually in the form of numbness, or numbness and tingling, accompany. The next most common mode of onset is impairment of vision, as a blurring or complete loss or a diplopia. Urinary symptoms such as urgency, hesitancy or precipitancy are also a common mode of onset. Other occasional onsets are vertigo, hemiplegia, dysarthria. These onset symptoms often improve after a few days or weeks.

In the well-developed case, the most constant findings on examination are evidences of *pyramidal tract disease* in the lower limbs, viz., weakness and hypertonus of one or both legs, hyperactive tendon reflexes, often clonus, positive Babinski reflex, bilaterally diminished or absent abdominal reflexes, and a spastic ataxic gait. A large number of cases have disturbed bladder-control as well. Due to the integrity of the anterior horn cells of the cord there is no muscular atrophy. Next in frequency are *paraesthesias,* chiefly in the limbs, such as numbness, tingling. About half of the cases show some slight objective sensory impairment mostly in the lower limbs.

Intention tremor, nystagmus, adiadochokinesis, dysmetric and hypotonia, due to *cerebellar involvement* occur in one or more limbs in more than half of the cases. Dysarthrias due to cerebellar involvement are quite common, but a true scanning (syllabic or staccato) speech is not.

Ocular Lesions: ocular palsies are seen in about 20%, due to lesions of the periaqueductal region of the midbrain. Anomalies of the pupil are rare, and the Argyll-Robertson pupil is very rare,

some denying that it is seen at all. The sclerotic plaques may develop at any point in the optic nerve or tract, but most frequently the axones from the retinal cells in the region of the muscula lutea, the center of distinct vision and the most sensitive to toxins and diffuse pressure, are involved. At first the inflammation in the plaque merely compresses the optic nerve fibres and interferes with their function, thus accounting for the ocular symptoms common at the onset of the disease. The result is the acute onset of defective vision in one or both eyes with one or more small central or paracentral scotomata. At the beginning the optic disc appears normal. But after an interval up to six weeks pallor of the disc usually appears and persists though the vision may be restored to normal after the acute onset. This disc pallor varies with extent and location of the plaques. There may be unilateral or bilateral optic atrophy; or merely a slight pallor of the whole disc; or merely a pallor of the temporal half of the disc due to involvement of the axones about the macula lutea. The last is the most frequent and the characteristic type of pallor found.

Conflicting reports appear in the literature regarding the *cerebro spinal fluid,* but the following statements may be made. There are no changes in the cerebro-spinal fluid pathognomonic of the disease. The spinal fluid pressure is normal, tending to be low rather than high. According to Hyland the C.S.F. is completely normal in 30-50% of the cases. The remainder show a small increase of lymphocytes (6-40 usually a count of over 50 being very rare), or of globulin, or both. The spinal fluid Wassermann is always negative. There is frequently an altered colloidal gold curve.

Some 50% of cases show abnormal colloidal gold curves of which 25% are of the paretic type. With the exception of epidemic encephalitis there is no other non-syphilitic condition in which a paratic type of curve may be found. In the absence of syphilis a paretic type of colloidal gold curve is of value in deciding whether a doubtful case is one of disseminated sclerosis. Failure to obtain such a curve is no evidence against the diagnosis, however. It has been stated that in the presence of a negative blood and spinal-fluid Wassermann, a paretic type of colloidal gold curve is most often indicative of disseminated sclerosis. An interesting point mentioned by Hyland is that occasionally during the course of the disease repeated spinal fluid examinations show the paretic curves tending to flatten out. No relationship seems to exist between the acuteness or severity of the clinical symptoms and the presence or type of colloidal gold curve.

The majority of cases show a *change of mood* in the direction of cheerfulness and happiness, and many show emotional instability. McAlpine remarks: "If a young adult, while telling you of a weakness in an arm or a leg seems extraordinarily cheerful about the whole affair, suspect disseminated sclerosis." There is a marked inconsistency between the patient's mood and his physical disability. A manic-like state may result. The rare case shows gross insanity. Emotional overaction in expression in the shape of laughing and smiling without adequate stimuli is common. A dense sclerosis of the periventricular region involving that part of the thalamus which borders on the third ventricle is a very common pathological finding, and it may be that release of thalamic centers from central control is responsible for the emotional overaction. Obviously this location of the lesion cannot be the sole cause. There is little doubt, however, that the mental manifestations are more dependent upon the organic brain disease than upon any constitutional factor. In contrast with these affective symptoms, intellectual disorders are minimal and negligible. There are no marked moral and ethical changes, such as are seen in paresis, though the cerebral cortex may be extensively involved by sclerotic plaques.

Course of the Disease.

The duration averages about eight years from the onset, but may run less

than a year or more than thirty years. A characteristic feature of disseminated sclerosis, seen in over 70% of the cases, is the remission. Every now and then men of experience begin to doubt the diagnosis they have made in a case of disseminated sclerosis because of the disappearance of many signs: the gait improves, the speech becomes less scanning, the spasticity is partially relaxed, the ataxic tremor decreases, and the patient reports great subjective improvement. The general improvement may be so marked that hysteria or other functional disease is suspected. The remission usually lasts for a few months. Some patients may have two or more remissions. Remissions are explained by the fact that the attack of the disease on the central nervous system strips the nerve fibres of their myelin sheaths producing temporary impairment of conduction. As the lesion subsides the function is partially or completely regained.

However each exacerbation leaves more permanent residual symptoms with the result that a slowly progressive disability usually results. Death in disseminated sclerosis is either from bulbar involvement, renal infections, bronchopneumonia or other intercurrent infections.

Diagnosis.

Because of the widespread dissemination of the lesions in disseminated sclerosis there is scarcely a syndrome which can not be produced by this disease. Isolated symptoms are of little value for diagnosis.

Charcot's classical triad-nystagmus, intention tremor, and scanning speech which is indicative only of involvement of the cerebellum, and which has figured so prominently in text-book descriptions of the disease, though pathognomonic when it does occur, is found in less than 10% of the cases. Many cases show one or two symptoms of this triad, but about one-quarter of the cases of disseminated sclerosis do not show a single component of the triad.

The most characteristic finding in disseminated sclerosis is evidence of pyramidal tract disease in the lower limbs associated with evidence of lesions elsewhere in the central nervous system. Perhaps the most helpful signs for early diagnosis are a slight nystagmus, a diminution of abdominal reflexes and a moderate spastic paraplegia. If there is a history of a discontinuance of the process in the early stages followed by a progressive course the nature of the disease can be determined with confidence.

Differential Diagnosis.

Encephalitis, Encephalomyelitis usually follows an acute infectious disease, there is fever, the course is more rapid, the patient is very ill, there is an absence of remissions, there is evidence of more diffuse invasions of the spinal cord substance including the anterior horn cells, there is a greater tendency towards a more fatal issue. Acute encephalitis lethargica is not a problem at present because of its great rarity. Schilder's disease may cause difficulty at the onset, but the patients are usually more severely ill, and the subsequent course of the disease determines its nature.

Cerebrospinal Syphilis: this disease may in the course of a few months develop a spastic weakness of the lower extremities with increases in the deep reflexes, and may show very marked periods of remission and exacerbation just like disseminated sclerosis. The positive blood and spinal-fluid Wassermann settles the diagnosis.

Cerebral Neoplasm: headache, vomiting, vertigo, optic disc changes (very rarely papilloedema) may occur at the onset of disseminated sclerosis, but there is generally a long interval elapsing between the onset of disturbances of vision and other signs suggesting disseminated sclerosis. Remissions and the subsequent course with the development of new symptoms indicating other foci serve to differentiate. Disseminated sclerosis is particularly apt to be confounded with a cerebellopontine angle tumour or a pure pontine tumour.

Multiple Areas of Softening of the Central Nervous System: diffuse arterial thrombosis in brain and cord may

give the syndrome of spasticity, tremor and disturbances of gait, and may show improvement later on. However, the age of the patient, the typical senile gait, and the rest of the clinical picture help to decide.

Other conditions rarely required to be differentiated are Chronic Internal Hydrocephalus, Cerebellar Atrophy, Pseudosclerosis, Tuberous Sclerosis.

Spinal Cord Tumours there is no cranial nerve involvement; superficial pain beginning at a definite segmental level and diminished superficial sensation or numbness below this level, and spinal tenderness opposite the affected segment are often loalizing signs; xanthochromia below the level of the block; double puncture with the Queckenstedt manoeuvre; lipiodol injection into the cisterna magna and X-ray of the spine—all may be required for diagnosis.

Syringomyelia: the sensory dissociation of pain and temperature, and the wasting of the small muscles of the hands make the diagnosis easy.

Subacute Combined Degeneration: the patient's age (45-60 usually), the absence of involvement of the cranial nerves, the normal C.S.F., the blood-picture either that of pernicious anaemia or the colour-index equal to or greater than 1, the absence of remissions, achlorhydria, the late appearance of sphincter trouble are in favour of subacute combined degeneration.

Tabes Dorsalis: a gradual onset, positive blood and C.S.F. Wassermann, Argyll-Robertson pupils, the normal plantar response, diminished knee and ankle-jerks, the normal abdominal reflexes, the slight loss of muscular power and absence of muscle are in favour of tabes.

Polyneuritis: the frequent presence of an obvious cause such as diabetes, alcohol, lead or arsenic; the absence of cranial nerve involvement; the later muscular wasting; the normal abdiminal reflexes, the absent plantar and ankle-jerks, the marked muscle tenderness are in favour of polyneuritis.

Friedreich's Ataxia: there is a distinct familial history, the abdominal reflexes are preserved, the tendon jerks decreased, and there are no remissions. Later there is a typical scoliosis and pes cavus.

Hysteria: the differentiation is particularly difficult when marked remission occurs. Important deciding factors are changes in the optic discs, diminution of abdominal reflexes, the slight sensory disturbances and mild urinary signs. Differentiation may require prolonged study.

Treatment.

In view of the different etiologies suggested, the most varied array of drugs has been used in treatment. In estimating the value of any drug, it must be remembered that the disease itself shows spontaneous remission. Silver arsphenamine, sodium cacodylate, mercury, various gold salts, quinine, malaria, typhoid vaccine, pilocarpine sweats, have all been tried with varying results. Diathermy has been useful in improving many. In general the treatment is symptomatic.

CANADIAN
LABORATORY
SUPPLIES
LIMITED

Canada's Leading Laboratory Supply House.

Headquarters in Canada for
LABORATORY APPARATUS
AND CHEMICAL REAGENTS

MONTREAL, QUE.
296 St. Paul St. West.

TORONTO-5, ONT.
Hartz Building. 32 Grenville Street

EPILEPSY — ITS CAUSES

By ANNE KEYL, B.Sc., '37.

Epilepsy was one of the first diseases described by ancient writers. We have references to and descriptions of seizures in the writings of the Greeks and Romans. Various causes have been assigned to epilepsy, and throughout history, there has been put forth a rather fascinating array. One of the most common and generally believed of these was that the epileptic was possessed by demons or evil spirits. Occasionally a more daring surgeon would cut holes in the skull to allow the demons to escape. Most of these unfortunate patients lost, not the demons, but their lives in the process.

Epilepsy in itself is not a disease, but a symptom of some underlying condition. In recent years, there has been a tendency among many clinicians to apply the term epilepsy only to those cases where no apparent organic defect can be found. This latter group has at times been called idopathic epilepsy.

In the past years, much valuable work has been done to determine the cause of epilepsy. Soon it became apparent that in some epileptics there was an organic defect, such as scars, produced by injury or disease or tumours. This has changed the general attitude toward epilepsy, with the result that these patients are now carefully investigated to establish the presence or absence of an organic lesion. However, there remains a large group in which examination is entirely negative. In this article, the alleged causes in this group will be briefly reviewed.

Heredity.

Until quite recently, the hereditary factor in epilepsy was greatly stressed. There seems to be no doubt that heredity plays some part, but not so much as was formerly thought.

Stanley Cobb gives some interesting figures in regard to heredity in epilepsy. The near relatives (parents, siblings and children) of a large group of epileptics and of a similar group of apparent normals, were studied in regard to the incidence of epilepsy. The epileptics chosen were non-institutional and non-traumatic cases. The incidence of epilepsy in the epileptic's relatives was 21/1000, as compared with 3/1000 of normals. Thus, epilepsy is some seven times more common among relatives of epileptics than among normals. Another interesting and significant fact was disclosed by this investigation. Among the relatives of epileptics, 43/100 suffered from migraine, while only 15/1000 of normals were in this class.

This investigation shows that there is an hereditary factor, but that it is not so great as was once thought. To the epileptic himself, this is of great importance. There is a common belief among the laity that epileptics should not marry and have children, for fear of transmitting the condition. However, from the above figures, one sees that the chances of this are not great. They should, however, not marry anyone with a family history of epilepsy.

Psychogenic Factor.

There seems to be a consensus of opinion that there is an underlying personality defect in epileptics—the so-called epileptic personality. Those who have studied them carefully, especially in epileptic colonies, describe them as egocentric, hypersensitive and secretive. They are rather intolerant and suspicious, and unable to get along well with their fellows.

L. P. Clark believes that this personality defect is the underlying factor. He says that these peculiar personalities are never properly adjusted to their environments and are unable to withstand the stress and strain of ordinary life. Their mal-adjustment is

well shown in the fact that many are unable to remain in one position for any length of time. They continually change their occupation, because it does not suit them, or they cannot get along with their fellow-workers. The seizures are the result of accumulation of stresses, and provide an escape from reality and responsibility by way of a period of unconsciousness. Clark believes that the treatment of epilepsy lies in the field of psychiatry.

It may be quite possible, however, that the disease itself causes the changes in character. These people live under a great handicap in our social system; they are continually afraid of an attack and finally stay at home and become seclusive and sensitive.

Cerebral Anoxaemia.

A considerable amount of work has been done on the theory that cerebral anoxaemia initiates the seizures in epilepsy. It is a well-known fact that cutting off the blood supply to the brain by tying the carotids in animals, or by strangulation in man, frequently produces convulsions. In carbon monoxide poisoning there is also a frequent occurrence of convulsions. Experiments have been performed to show the effect of oxygen deprivation in epileptics and in normals. The epileptics show symptoms sooner than normals, and frequently react by having seizures.

Surgeons, when operating on brains, have noticed constriction of pial vessels preceding convulsions. Vaso-constriction has been noticed elsewhere in the body before seizures, such as pallor of the face and constriction of retinal vessels.

Until recently, the cerebral vessels were not thought to be under vaso-motor control, but Penfield and others have demonstrated that they have a sympathetic supply, capable of producing vaso-constriction. Hence the convulsions of epilepsy may be due to an anoxaemia produced by vaso-constriction of cerebral vessels.

Toxic Factor.

Cuneo and Reid are the authors of this theory. They believe that there is a toxic substance produced which, in entering the circulation, affects the brain, producing certain changes. Cuneo demonstrated such a toxic substance in the blood of epileptics soon after a seizure. It was identified as a proteose. Also, when certain proteoses are injected into the blood stream of animals, convulsions occur.

Proteoses are produced in the gastro-intestinal tract as a result of protein digestion, but normally do not gain entrance to the circulation. From these facts, they thought that if epileptics were put on a high protein diet they would become worse. Therefore, one group was given a high protein diet and another group a high carbohydrate diet, expecting the first group to show an increased incidence of epileptic seizures. However, the reverse occurred—those on the high protein diet improved while the others became worse. Thus, the gastro-intestinal tract and protein metabolism were not at fault. These workers are still busy on this problem.

Toxic substances certainly do cause convulsions, for one sees them in such conditions as the acute exanthemata and eclampsia. It is possible that epileptic convulsions are caused by some toxic substance produced as a result of some derangement in metabolism.

Increased Intracranial Pressure.

Increased intracranial pressure has come in for its share of attention as a cause of epilepsy. In acute meningitis, where intracranial pressure is increased, convulsions do occur, but they also occur when the pressure is low. Studies of cerebro-spinal fluid pressures have frequently been made in epileptics and the majority of these are normal. Lennox and Cobb found the following results in 400 epileptics:

71%—C.S.F. pressure normal.
17%—C.S.F. pressure 200-250 mm.
8%—C.S.F. pressure below 100 mm.

So Suitable for Giving !

THE gift of Sterling Silver Dresserware will prove a very happy solution to your gift problem. When you choose Vanity Sterling you present a gift of immediate importance and lasting beauty. Many beautiful patterns may now be seen at Birks-Ellis-Ryrie.

The Astrid (as illustrated) Mirror, Brush and Comb **$45.**

BIRKS-ELLIS-RYRIE

YOUNG AT TEMPERANCE — TORONTO

Freemont-Smith and Lennox also studied the variations in cerebro-spinal fluid pressure in two cases, in relation to fluid intake. The pressures were taken after one period of fluid restriction and after another of excessive fluid intake, and showed only small variations from the normal pressures. Pressures were also read continuously in these patients while they were taking large amounts of water. Little or no variation was found. This shows that the majority of epileptics have normal cerebro-spinal fluid pressures, and that fluid intake has no effect on it.

Yet, the fluid intake has some effect on epileptics, for when they are on a restricted fluid regime they have fewer convulsions. One explanation offered is that there is an oedema of cerebral tissue producing the convulsions, and the absence of fluid required to cause this, reduces the number of attacks.

Destroyed Association Pathways.

Convulsions have frequently been noted months and years after head injuries. In these cases, there is frequently a gliosis and scar formation, which may act as an irritant to the nerve cells, causing abnormal discharges. Then again, the scars may interrupt the axis cylinders, producing gaps in association pathways. Thus afferent impulses coming up may be short-circuited into efferent paths, setting up an explosive response. In epilepsy, there may be such an interruption of association paths, and afferent impulses are shunted into many wrong efferent paths.

From the variety of theories presented, it is seen that our knowledge of the underlying factors of epilepsy are as yet little understood. However, the amount of experimental work done on the problem has put the treatment on a much more sane and humane basis.

A discussion of the treatment will follow in the next issue of this journal.

Bibliography.

Stanley Cobb, Arch. Neurol. & Psychiat. 1932, vol. 28.

Michael Osnato, Arch. Neurol. & Psychiat. 1932, vol. 9.

The writer wishes to acknowledge the interest and aid received from Doctor H. Hyland in the preparation of this paper.

EMPYEMA — A REVIEW OF ITS ETIOLOGY, DIAGNOSIS AND TREATMENT.

By A. D. RICE, B.A., '37.

Introduction.

As its name implies, empyema signifies pus within a natural cavity, but usage has so limited its application to pyogenic affections of the pleural space that one hardly needs to apologize for limiting this article to a consideration of empyema thoracis.

History.

Empyema was recognized by Hippocrates, who realized that it was a frequent complication of pneumonia. He described its symptomatology, treatment and prognosis. It is interesting to note that he advised thoracotomy, with either knife or cautery, to permit free drainage, daily dressing and the injection of oil and wine into the cavity.

Subsequent advances have been made, but alterations in the details of technique — flap-like incisions, open drainage, and various methods of closed drainage, either by repeated aspirations or some form of continuous, airtight suction. Until 1918, when the Empyema Commission of the United States Army published its findings, no major improvement had been made on the treatment advised by Hippocrates. This Commission was the first to formulate the principle that an open pneumothorax is to be avoided in the formative stages of empyema. They advocated repeated aspirations until frank pus had formed and the process was walled off, when an open type of drainage, following rib resection, should be instituted.

Etiology.

Suppurative pleuritis is rarely, if ever, a primary condition. In the vast majority of cases, it is the result of extension of an infective process from the lungs, especially a lobar or bronchopneumonia. Other pulmonary sources include abscess, gangrene, new growths, septic infarcts, bronchiectasis, and tuberculosis. Empyema may develop in association with mediastinal lesions, such as ulcerating cancer of the oesophagus, or may result from a primary condition in the chest wall, as gunshot wounds, stabs or fractured ribs. Extra thoracic sources are the acute exanthemata, tonsilitis, pharyngitis and sinusitis, or the infection may extend upward through the diaphragm from a subphrenic or hepatic abscess. Infection of the pleura may also take place from the blood stream from a primary focus anywhere in the body.

Bacteriology.

The micro-organisms most frequently encountered are the pneumococcus and the streptococcus hemolyticus. Since the pneumococcus is the most common cause of pneumonia, this organism is most frequently found in the suppurative pleuritis, which arises as a complication. However, in the epidemic pneumonia, following measles and influenza, which occurred in 1918 and 1919,streptococcus hemolyticus was twice as common as the pneumococcus. Brooks and Cecil, in their study of empyema during this epidemic, found that pneumonia due to streptococcus hemolyticus, was followed by empyema in 36% of the cases, while pneumonia due to the pneumococcus, resulted in empyema in only 11% of those affected. During a non-epidemic period, Lord found in a study of 137 cases of empyema that the pneumococcus was responsible for the infection in approximately 40% of the cases, and streptococcus in 20%.

Any of the pyogenic bacteria may cause empyema. Among the more rare offenders are staphylococcus, colon bacillus, B. influenzae, B. typhosus and

others. Fungus infections of the lung, such as actinomycosis and blastomycosis may be followed by empyema.

Virulent bacteria can gain entry to an alveolus from the bronchial tree, as in a pneumonic process, and then it is doubtless easy for the inflammation to spread from a peripheral alveolus to the adhering layer of pleura. This is presumably the mode in which a pneuococcic empyema develops.

If virulent bacteria gain access to the peribronchial tissues, they can spread directly to the pleura through the interstitial lymphatics and tissue spaces. This type of empyema presupposes a lobular or bronchial pneumonia and the infecting agent is most likely to be a streptococcus.

Pathology.

Like other serous membranes, the pleura is covered by a single layer of flattened nucleated cells, united at their edges by cement substance. These cells rest on a basement membrane. Beneath the basement membrane, there are networks of yellow elastic and white fibres imbedded in a ground substance which also contains connective tissue cells. It is in this subendothelial layer that the blood vessels, lymphatics and nerves of the pleura run.

The essential feature of empyema is the presence of pus in the pleural cavity. It constitutes, therefore, an abscess in the pleural space. Marked variation in the exudate is encountered, from a cubic centimetre to several litres. It is more common to find a large amount of exudate in the steptococcal empyema than in a pneumococcal empyema. In the interstitial peribronchial inflammation, which occurs with the streptococcus, the points of attack on the visceral pleura are likely to be more numerous and spread over a greater area than in the case of pneumococcal pneumonia, which is generally confined to one lobe. In the latter condition, the fibrous adhesions form early and tend to keep the pus confined to the region immediately adjacent to the affected lobe.

In the early or formative stages of an empyema, the pleural effusion may be serofibrinous, blood-tinged or distinctly hemorrhagic. Such fluids are noted especially in the early stages of an empyema due to the streptococcus, and even in an early stage, there are numerous organisms in the exudate. MacCallum describes the fluid exudate in streptococcal cases as follows: "Numerous epithets have been applied to it—it is like pea soup, like turbid urine, like unstrained bouillon, like muddy water, but generally like thin pea soup." It often has a greenish cast and the granular sediment which can be stirred up in a cloud, tends to settle in the bottom of the pleura. Later it becomes more thick and turbid, quickly assuming a more purulent character, while the exudate due to pneumococci is much earlier found to be highly fibrinous and a thick yellow pus. The pus from a case of empyema due to colon bacilli, will be markedly foetid, while a brownish discolouration suggests blood and is more usual in tubercular empyema.

Microscopically, polymorphonuclear leukocytes are the most important constituents, and, in addition, a variable number of erythrocytes, degenerated endothelium, cellular debris, cholestrin crystals and bacteria may be found. Sterile purulent exudates are usually tuberculosis.

Incidence.

Empyema is more common in children as a sequel to pneumonia than it is in adults. In the latter, the incidence is greatest between the ages of twenty and forty. Men are more frequently affected than women, due to the greater frequency in males of the underlying condition. In children, the distribution between the sexes is approximately equal.

Penberthy and Benson, in a study of 407 cases of empyema at the Children's Hospital, Detroit, for the ten-year period from 1926 to 1936, found the highest incidence at two years, with a slow fall in the number of cases to eight years, with only a few cases in the age group from ten to twelve years. They

also showed that, in their series, the percentage mortality was highest in infants under one year, dropped rapidly to about twelve per cent. in those from two to eight years of age, and more slowly to a very small percentage in those over nine years of age.

Symptoms.

The onset may be acute and resemble serofibrinous pleurisy, the differentiation being made by thoracentesis. But in the majority of cases, the onset is insidious and obscured by the symptoms of the primary disease. An intensification of the symptoms of the primary affection (lung abscess), a prolongation of the symptoms beyond the time when they should clear (pneumonia), or the recurrence of symptoms in convalescence from a respiratory infection may characterize the onset of empyema. Cough may be distressing and dyspnoea of varying intensity is usually present. As a complication of pneumonia, empyema may develop as early as the third day or as late as the fourth week after defervescence. The general symptoms, as a rule, are quite distinct — chilliness, irregular fever, rapid pulse, sweats, increasing pallor, and leukocytosis. The leukocyte count varies from 15,000 to 40,000, the increase being due largely to polymorphonuclear cells.

The streptococcic cases are the most severe and the most rapidly fatal; the pneumococcal types are not infrequently mild and respond most favourably to treatment; while the tuberculous type tends to chronicity with less marked general symptoms.

Physical Signs.

The physical signs are those of fluid in the chest, but the presence of previous or still existing consolidation may make it difficult to recognize. An increase in the feeling of resistance, with flatness instead of dullness on percussion, disappearance of tactile fremitus, diminution of breath sounds and absence of râles are characteristic findings. It is important to remember, however, that loud bronchial breathing may be present, even in the presence of fluid, especially in children, and when the lung is markedly collapsed and adjacent to an unobstructed bronchus.

The purulent material may become encapsulated between the fissures of the lung or anywhere in the pleural cavity. The physical signs of interlobar or encapsulated empyema may be slight, but a search over the lung fissures for an area of slight dullness and diminished breath sounds should be made in all cases where the symptoms of empyema are found without the signs of gross pleural exudate.

A useful method of distinguishing between consolidation and fluid is to percuss out the mediastinum. If it is in the usual position, one thinks of consolidation, whereas fluid will cause the mediastinum to be drawn away from the affected side.

A fluoroscopic examination is of great value in noting the shadow cast by the fluid and in ascertaining whether the mediastinum has been shifted. X-ray plates offer one of the best methods of detecting interlobar collections of pus.

Clubbing of the fingers is a late sign, but it is said that the experienced eye can detect the early changes by the second or third week of the disease.

The above signs furnish presumptive evidence of empyema, which can only be definitely proven by aspiration of purulent material from the pleural cavity.

One should be careful not to place too much dependence upon physical signs, as a thin layer of fluid over an area of consolidation may allow bronchial breathing and tactile fremitus to be manifest. Careful and experienced observers have at times been unable to distinguish pleural effusion from solid lung, finding increased breath sounds in the former and often decreased breath sounds in consolidation.

Thus the four cardinal points, upon which a diagnosis of empyema is made, are: (1) The patient's fever increases and his general condition becomes less

satisfactory than one would expect, considering the uncomplicated primary disease; (2) the physical signs of fluid in the chest are elicited, this is the least reliable point; (3) the roentgenogram shows the shadow of fluid, it may be encapsulated and interlobular; (4) purulent fluid is obtained on aspiration.

The differential diagnosis rests between empyema and lobar pneumonia, chronic fibrinous pleurisy, massive collapse of the lung, thoracic tumours, pericardial effusion, and abdominal disease, such as subdiaphragmatic or hepatic abscess. In empyema with a large effusion, there is displacement of the heart to the opposite side, whereas in the conditions mentioned, the heart is not displaced, or, as in collapse of the lung, drawn over to the same side. Labor pneumonia is usually sudden in onset with a chill, high fever, dullness rather than flatness, the percussion changes corresponding to a lobe, bronchial breathing, bronchophony, high-pitched, crackling râles, rusty sputum, herpes and the absence of displacement phenomena. In chronic inflammatory thickening of the pleura, the physical signs are similar to those found in empyema. The retraction of the chest wall, displacement of the heart toward the affected side, the absence of clear-cut definition of the upper limit of flatness, and the association of evidences of pulmonary fibrosis enable one to make the differentiation.

Primary intrathoracic tumours may be differentiated by a careful study of the location and extent of the dullness, a search for extrathoracic metastases, especially in the cervical nodes, the location of the mediastinum and the roentgenogram.

While large pericardial effusions may stimulate left-sided pleural effusions, the former are accompanied by more marked dyspnoea absence or enfeeblement of the cardiac impulse, with displacement of the heart upwards rather than to the right, flatness corresponding to the shape and extent of the distended pericardium, and the X-ray appearance of a shadow corresponding to the pericardium.

Prognosis.

The prognosis varies with certain factors. First, the age, the mortality being about 75% in children under two years of age and in the very old. The invading organism also has a great deal to do with the ultimate outcome, other factors being equal, the mortality in streptococcal empyemata is several times that of pneumococcal empyema. The mortality rate also varies in different years, even when the patients are given the same treatment by the same staff. This seems to depend upon the virulence of the underlying pneumonia. Treatment is perhaps the most important factor when considering prognosis, as it, at least, may be altered to reduce the mortality. As an example of this, one might quote from the Surgeon-General's report with regard to the mortality in Camp Lee from empyema. During the early part of the war, when the cases of empyema were treated by early operation, the mortality was 40%. This fell to 4.3%, when aspiration with deferred drainage operation was substituted.

Thus the best prognosis may be given in those cases treated with early aspiration and operation at the optimal time with the avoidance of an open pneumothorax. Untreated acute cases go on to chronicity and may terminate by gradual absorption and deposition of lime salts, by perforation into the lung and discharge through a bronchus, or by perforation of the chest wall, empyema necessitas.

Complications.

Space will only permit a brief summary of the more common complications. These are: Rupture of the empyema into the lung with bronchial fistula, rupture of the empyema through the chest wall to discharge externally, the development of a suppurative pericarditis, peritonitis, bacteraemia and septicaemia, development of a bilateral empyema, mediastinal abscess, and other miscellaneous complications such as meningitis, brain abscess and multiple arthritis.

Treatment.

Those who are familiar with the literature are aware of the numerous methods of treatment advocated by various authors, most of which differ only in their more minute details. As a discussion of these various methods is outside the scope of this review, we will confine the discussion to certain broad principles in treatment with, perhaps, a short account of one or two of the more popular techniques.

In its broadest application the principles of treatment to be followed are: (1) to save the life of the patient, and (2) to prevent chronicity. Most authorities are agreed that the patient rarely dies of his early acute empyema. He may succumb to the underlying disease, or to ill-advised operations devised to relieve the empyema in the early stages. Or if he survives for several weeks and his empyema is improperly treated, or not treated, he is likely to fall victim to some intercurrent infection to which his weakened condition makes him a ready prey.

"Free evacuation of pus wherever it occurs" is one of the established principles of surgery, and in empyema holds good with certain distinct reservations as to the method of drainage. The principle expressed by the phrase "primum non nocere" must ever be kept in mind, for one must not in one's zeal to get free drainage do anything that will further embaress an already embarassed cardio-respiratory system. While a healthy individual possesses a consideration cardio-respiratory reserve as indicated by his vital capacity, this is not the case in patients with acute empyema. Here, through pneumonic consolidation, a considerable portion of the lung may be thrown out of function, fluid accumulations in the pleura may be compressing other areas and cardiac action is compromised by the changed pressure relations within the chest and by the severe toxaemia. The high fever and consequently elevated metabolic rate cause increased oxygen requirements, which throw an added load upon an already embarassed cardiorespiratory mechanism.

In such cases it is imperative that one shall institute a type of treatment which will throw as little additional strain upon cardio-respiratory physiology as possible. An operation such as immediate rib resection which produces an open pneumothorax is therefore contra-indicated. Either of two procedures remains: (a) repeated aspirations or (b) airtight, continuous suction drainage.

Repeated aspiration was the method recommended by the Empyema Commission during the war. Its object is to withdraw the pus without producing pneumothorax, thereby correcting to a degree the abnormal pressure relationships within the chest, diminishing the toxaemia and improving the general condition of the patient. This should be repeated every day or two, or as soon as sufficient effusion recurrs to make it necessary.

But when the formative period is past and frank pus is aspirated, there is comparatively little danger from an operation to procure open drainage for the following reasons: (1) there is less danger of creating a pneumothorax as the pus is well walled off by adhesions; (2) even if open pneumothorax is created, its harmful effects are lessened because the mediastinum will be less mobile due to adhesions, the vital capacity will have increased, the patient's need for oxygen will be less as his metabolic rate decreases, and the accessory muscles of respiration will not tire as rapidly as they would have at the height of the disease with its general toxaemia. (3) the patient will be in better condition to withstand the shock of the necessary surgical procedure.

The rare case may clear up with repeated aspirations, but the majority will require a more efficient type of drainage before the cavity will be obliterated.

Continuous closed drainage is advocated by some as the best method of treatment. If it works there is no doubt that it possesses the advantage

of simplicity of procedure, for it can be instituted early under local anaesthesia, is not harmful to the patient, and avoids the necessity of a later operation to procure open drainage. The critics of this method claim that only a small drainage tube can be used as the connections must remain airtight, and consequently the tube will rapidly become plugged necessitating the operation, to avoid which, this technique was instigated. On the other hand the advocates of closed drainage, notably Bettman of Chicago, claim that with reasonable care the tube need never be plugged and bring forward a series of cases in which this method alone was used with success in a very large percentage of the cases. Bettmah claims that those who fail in this method of treatment do so because they do not adhere strictly to the careful details of his technique.

If the technique of early aspirations and deferred open drainage is used one has several choices as to the type of open drainage to use. One of the most usual operations is rib resection, which is done under local anaesthesia. The essentials of this operation are the incision in the periosteum and the removal of a small section of rib, wide incision in pleura and some adequate means to keep the wound open till the cavity is obliterated. The critics of this method point to the frequency of osteomyelitis in the cut ends of rib, and the possibility of small chips of bone falling into the empyema cavity, either at the time of operation or later when they become separated from the rib due to inflammatory processes. These sequestra are troublesome and are frequently the cause of persistent discharge in old empyema which have done well in other respects. Such conditions led the Empyema Commission to advocate a simple intercostal drainage operation. If in a particular case it was found necessary to enlarge the opening, this could be done by subsequent rib resection. Usually, however, simple thoracostomy is sufficient.

Yet in spite of the fiery debates that have taken place over the relative merits of rib resection as opposed to simple thoracostomy, the question is not of vital importance provided neither operation is performed until the pneumonic process has subsided.

In a few cases some sort of plastic operation will be necessary in order to procure collapse of the cavity. But these are the rare cases, those that have not been diagnosed early or that have gone on to a chronic type of empyema due to inadequate treatment.

Those cases, in which the infecting organisms are of more rare type, are treated in the same manner as are patients infected with the commoner pyogenic organisms, with the addition of specific treatment where such exists.

In all cases of acute empyema in addition to the local treatment outlined above there are certain other considerations of almost equal importance. These consist of the general care of the patient, good nursing, sunlight, fresh air, and a nourishing diet. Evarts Graham is particularly insistent upon the last point. He points out that due to the fever and the toxic process there is a raised metabolic rate with increased protein requirement. Because this is frequently not realized the patient develops a negative nitrogen balance and is forced to use up his own tissues.

Breathing exercises, such as blowing coloured water from one bottle to another, are frequently of use in expanding the lungs and thus forcing the purulent matter out of the chest and collapsing the cavity.

If after careful treatment a non-tuberculous empyema fails to heal and show signs of spontaneous closure in a reasonable length of time the fault likely lies with the surgeon, and one of the following is almost certainly the cause of his failure: Inadequate drainage, or the presence of a foreign body in the cavity.

Tuberculous Empyema.

Because this subject is an involved and controversial one, it is merely mentioned here that this review may be complete. One cannot, in a paper of this length, go into the various opinions

held by different authorities, but may only outline the more generally accepted principles in the treatment of this type of empyema. As this condition is practically always associated with tuberculosis the usual treatment for that condition should be instituted, and is of paramount importance. The tuberculous empyema is best drained by repeated aspiration, and some cases have apparently cleared up under this régime. Open drainage is contra-indicated in the opinion of many authorities, because of the very grave danger of a chronically discharging, tuberculous sinus with the ever present danger of superimposed secondary infection. The tuberculous patient who happens to have a non-tuberculous empyema is treated as outlined for non-tuberculous cases.

Summary.

Due to the premium on space, a rather brief and sketchy review of empyema has been presented. An attempt was made to emphasize the essentials in the diagnosis of such cases: (1) an increased severity in the symptoms of a patient with a respiratory tract infection which are not warranted by the usual course of the primary disease; (2) the signs of fluid in the chest, this is the least necessary of the four essentials; (3) the visualization by X-ray of the effusion and displacement of the mediastinum away from the affected side; (4) definite proof by aspiration, microscopic examination and culture.

Once the diagnosis has been established one of the several definite types of therapy may be followed, based on proven principles. For it does not seem to matter what the infecting organism is, with the exception of the tubercle bacillus, if the virulence of the bacteria is not high enough to kill the patient early, he will get better promptly once an adequate form of treatment is instigated.

We have endeavoured to show that the essentials of adequate treatment consist of: (1) avoidance of open pneumothorax in the formative stage with consequent respiratory embarrassment, (2) avoidance of chronic empyema by the rapid drainage, sterilization and collapse of the cavity, and (3) careful attention to the patient and his needs, particularly his nutritional requirements.

It was also pointed out that the principal means of procuring obliteration of the cavity — (a) early aspiration and deferred open drainage, and (b) early, continuous, closed drainage —were both basically sound and in proper hands would yield excellent results.

Bibliography.

1. Bettman—Am. J. Surg. March, 1936.
2. Travers—Am. J. Surg Oct., 1936.
3. Pentberthy and Benson—Annals of Surg. Oct., 1936.
4. Funk — Oxford Monograph Series—Chronic Diseases of Lungs.
5. Graham—Gross Prize Essay—"Empyema Thoracis."
6. Andrus—Pract. Lib. Vol. IV.
7. Barach—Pract. Lib. Vol. III.
8. Graham, Singer and Ballon—Surg. Diseases of the Chest.

The J. F. HARTZ CO., Limited

Carry a complete line of

MEDICAL STUDENTS' SUPPLIES

STETHOSCOPES, OTOLARYNGEAL SETS,
HAEMOCYTOMETERS, HAEMOGLOBINOMETERS.
STUDENTS' DISSECTING SETS

34 GRENVILLE STREET TORONTO, CANADA

LEAD POISONING IN THE AUTOMOBILE INDUSTRY.

PART II.*

Laboratory Aids to Diagnosis; Theoretical Aspects.

By ALASTAIR MACDONALD, '37.

The previous article* will have made the reader familiar with the general aspects of the situation. It is now proposed that we discuss in more intimate detail the laboratory methods which were used in reaching a diagnosis. Some of these are new and have only recently come to the fore.

As an introduction, a summary of the findings of the air samples will be of interest. Samples were taken in July, 1935, of the air which the disc-sanders were breathing. This was done by drawing the air through a water bottle and metering the flow. The impinger or intake was held close to the man's head while he was carrying on his work. The lead trapped in the water was extracted as chromate of lead, and the amount determined. The results, as shown below, indicate the high exposure of the workers.

Discing with mouth of impinger up, 1260.0 mg. lead/10 cu. m. air.

Discing with mouth of impinger down, 375.0 mg. lead/10 cu. m. air.

This indicated a rather rapid fall of the particles, presumably the coarser ones, but a concentration anywhere near the lower figure constitutes a major hazard! A test, run for forty minutes, commencing five minutes after quitting-time, showed a concentration of only 3.3 mg. lead per 10 cu. m. of air. This still indicates a rapid fall of particles, but it must be remembered that a concentration of over 2.0 mg./10 cu. m. is not considered safe.

In order to determine the efficiency of the respirators, samples of the materials used were placed over the impinger and air from about the discing operation was drawn through them.

The following are the results of this procedure:

Flat type of mask (1 layer thick), 19.0 mg./10 cu. m. air.

Bag type (used many times), 7.0 mg./10 cu. m. air.

New bag, 1.153 mg./10 cu. m. air.

Washed bag (several times), 5.79 mg./10 cu. m. air.

It is sen that the flat type of mask with only one layer of material was grossly inefficient, as it allowed 19.0 mg. of lead/10 cu. m. air to pass through. The bag type, when washed many times, was also inefficient, but those that had been washed once or twice were found to be fairly efficient. In the previous article, we pointed out that the flat mask, with several layers of material, was found to be the most satisfactory. While we have not the exact figures to prove this point, we wish to assure the reader that this is true. There is also a fallacy which is not apparent from the figures. In testing the bag type, no consideration is made for the dust which settles through the open mouth of the bag while it is hanging about the man's neck between operations. Considering all points, the flat mask is the better. It is to be noted that, in testing, the air is drawn through the masks at a rate approximately that of a normal inspiration of a man doing moderate labouring work.

The next consideration is that of diagnosis. We are given a condition of exposure and we cannot wait till clinical symptoms appear, in order to reach a decision. We have two possible aids in determining whether or not a

---

* Lead Poisoning in the Automobile Industry, Part I., Vol. XIII., No. 4, February, 1936, by the same author.

man is absorbing lead in dangerous amounts—urinalysis and examination of the blood smear. When the work was first attempted on a large scale in the automobile factories, urinalysis was tried, but found to be inaccurate and cumbersome, due to the method employed. Since that time, a new, simpler and accurate method has been devised by the laboratory staff of the Sick Children's Hospital in Toronto, which is deemed to be very satisfactory. However, in the instance under discussion, the situation demanded even simpler and speedier technique, due to the large number of examinations required. The blood smear was, therefore, resorted to, as a method of diagnosis.

The Blood Smear.

There are several methods of staining the blood smear for stippling or reticulation, all of which involve the use of a rather alkaline stain, since the material forming the stipples is intensely basophilic. A neutral or acid stain is worthless here, as the punctate basophilia will be only very poorly demonstrated at best. It is thus seen that the employment of a polychromatic stain, such as Wright's or Hasting's, is practically useless. Very little, if any, stippling will be demonstrated, even in moderately severe cases of plumbism, and that which does show up is of such a fine, showery nature and neutral colour, that it is very easily missed. The stains usually employed are rather alkaline aqueous stains of about pH8. With the ordinary stains for stippling, the smears are spread in the usual manner and fixed by immersion in absolute methyl alcohol for about five minutes, then they are immersed in jars of stain. It is to be noted that in no case is the stain poured onto the slide; the slides are immersed in the stain, as dust and precipitated material would otherwise settle out and confuse the picture. The chief ingredient of these stains is methylene blue with sodium or potassium citrate and carbonate. Potassium is believed to favour the staining of the basophilia more than sodium salts.

The stained smears, on examination, appear to be monotones of various shades of blue or green. The stippling or basophilia, as it is often termed, show up as an intense blue-black dotting scattered throughout the red cells, usually more densely collected about the periphery. The granules vary in size from those seen in the basophilic leucocytes, which are quite large and dark, to those approaching the fine granularity of the neutrophilic cytoplasm.

Some authorities maintain that the size of the granules is of diagnostic importance, the larger granules being of much greater significance in plumbism than the smaller ones. It is considered that a relatively large number of finely stippled cells is of little consequence, as compared with a lesser amount of coarser stippling. It is the fine variety of stippling which is most frequently seen in pernicious anaemia. The large granules are certainly much more frequently encountered in plumbism, and one cell does not seem to contain a mixture of the large and small types. Very little, if any, variation in size of the granules can be determined within a cell. It cannot be too strongly pointed out that the presence of stippled cells is in itself not diagnostic of plumbism. There must be at least 5,000 or more stippled cells per million red cells before a diognosis of lead absorption or plumbism can be made. This calls for an accurate estimation of the number of normal red cells in proportion to the number of stippled cells, which is done by counting the number of each type of cell seen in the microscopic field until a representative figure is obtained. Usually 15 to 20 fields should be sufficient.

Other features of the smear are the presence of polychromatic cells—cells which seem to be off colour, so to speak, and are considered by some to be the precursors of the stippled cell. These cells, however, are best seen with a polychrome stain. There also seems to be present, from casual observation, a slight to moderate lencopoenia in the more severe cases, and in many of these, normoblasts can be seen. These occur

in numbers from about 0.1% to 0.3% and are only encountered when the stippled cell count, and more particularly the reticulocyte count, is rather high. Stippled cell counts upwards of 3.0% or 4.0% are not uncommon in acute cases of plumbism.

The McCord Method.

The foregoing is an account of the commonly accepted methods of examination for stippling. We now propose to introduce a new, and in some respects, unusual method of blood examination, which has been embraced by the automobile industry, with a fair degree of satisfaction. It is the method which was propounded and employed by McCord in his extensive work with lead poisoning.

A thin, even smear is made in the usual way and allowed to dry from one to three hours. The author of the method maintains that the drying is an essential part of the process, since, if it is not properly done, some of the material is liable to be lost from the smear in the staining process. If the drying is too long, the staining properties of the cells are liable to be altered. The optimum length of time depends on temperature and relative humidity, and can only be gauged in the light of some experience and experimenting.

The smear is now fixed in the following manner: A strip of filter paper is laid lengthwise along the slide in such a manner that it covers half its breadth. Absolute methyl alcohol is carefully run over the paper until it is thoroughly moistened. The paper must not be flooded, for the alcohol is liable to run over to the other half of the smear, which must remain unfixed. The alcohol dries in a few minutes; the paper curls up and can be blown off. We now have half the smear fixed and half unfixed, with the line of division running down the *long* axis of the smear. The slide is now immersed in McCord's stain which is a modified Manson's stain and left from five to ten minutes. A readable stain can be obtained in as short a time as two minutes. It is practically impossible to overstain, even though the slide be immersed for very long periods of time. It is important that the stain should *not* be kept in stock bottles, but should be made up and immediately poured into the Coplin jars which are used for straining. This is done due to the fact that a great deal of annoying precipitate will form and mask the smears so greatly as to make them useless. The chief source of the precipitate is a fine film of oxidized material which forms on the surface of the stain, and should the liquid be kept in stock bottles this film is finely pulverized if the stain is poured out into jars. When the stain is kept in the jars the film may be removed before using, by scooping it up with the end of a glass slide. This caused some considerable trouble until the difficulty was appreciated. Our associates in Detroit were making up stain every few days in order to avoid the resulting precipitate. Fortunately the stain is quite cheap. Stain kept in the above manner will last several weeks before evaporation requires that it be discarded. Climatic conditions certainly have a very pronounced effect on the rate of formation of the oxide film. The composition of the stain is as follows:

Medicinal methylene blue (Merck), 2 grams.
Sodium borax, 0.4 to 0.5 grams.
Boiling distilled water, 100 c.c.

Dissolve the borax in the boiling water and then add the methylene blue, stirring well until it is all dissolved. Do *not* boil the solution after adding the methylene blue. Pass the stain through a fine filter paper four or fine times and allow it to cool to room temperature. No ageing is required. The stain is ready for immediate use. It is to be noted that this is an aqueous stain; there is no fixative in it. Fixation of the smear is as previously desired.

Examination of the Smear and Interperetation.

Since the stain is aqueous, the cells on the unfixed side will be laked. All that is seen on examination is the very dim outline of the ruptured cell membranes, but it is here, however, that

our anticipated configuration appears. McCord aptly terms it "basophilic aggregation." It is the result of the flocking together of the basophilic material in each pathological cell. It appears as a cross between stippling and reticulation, often having a sort of clock-face outline. A heavier condensation of basophilic material (chromatin?) appears about the periphery with diffuse stringy threads running tortuously across it having the punctate dots like stippling scattered about here and there. This picture is easily seen due to the beautifully sharp contrast offered by the deep blue staining configuration against the clear, glass-like background. The only items which stain up on the unfixed side of the smear are the basophilic aggregates and the leucocytes.

The following is the method of estimating the number of aggregates and determining their ratio to the normal cells. On the unfixed side of the smear twenty fields are examined and the total number of aggregates is recorded. Moving up to the fixed side of the smear where all the blood cells stain an even blue, we count the total number of red cells seen in five fields, spotted at approximately equal distances apart and parallel to the area counted on the unfixed side. The smears must be carefully spread in order to be sure of even distribution of the red cells. By a simple arithmetical procedure we may thus obtain the percentage of aggregates present which is the most convenient method of recording the findings. It has been found that when the aggregate count lies between 1.25% and 1.50% we are on the border line of normalcy. Figures above this level are taken to indicate a definite pathological condition; in these cases the absorption of lead is beyond safe limits. Clinical symptoms do not usually begin to appear until the count is from 3.0% to 5.0% or higher, but no definite rule can be laid laid down as exceptions have occurred. It has also been noticed that benzol and carbon tetrachloride poisoning may give rise to a similar picture.

Leaving the aggregate for a moment, let us consider the fixed side of the smear which was used to estimate the number of red cells. Many useful hints may be obtained if we glance over this area before looking for aggregates. All the cells stain an even blue, the intensity of which depends on the amount of haemoglobin which is within the cell. The slightest abnormalities of morphology and staining properties are readily discernible; smears stained with the ordinary polychrome stain seem dull and indistinct by comparison. Any evidence of anaemia is easily seen. Normoblasts stain particularly well, the central nuclear material taking on intense blue black colour and the surrounding cytoplasm a higher blue. Polychromatic cells can occasionally be made out, but since no acidophilic stain is employed a proper differentiation cannot be made. No stippling or aggregation is seen here, due to the deepness of the stain.

The leucocytes also stain very well, the nuclei taking on very dark hues, and the cytoplasm very pale, washed-out shades. The morphological characteristics of these cells are so admirably portrayed by this staining process that it is very easy to accurately differentiate all types. It was previously stated that a slight to moderate leucopoenia is encountered in the more severe cases. I find that this is mainly a reduction of the number of granular leucocytes. This point may be of some theoretical significance and will be mentioned when we consider that aspect of the situation.

The results of the examination of the blood smears for aggregation is shown in the following table (page 74). Over 550 smears were examined, the bulk of which are included.

It will be observed that the greater proportions appear in the groups below 0.60%, with the exception of the Toronto Branch reports for July, where the maximum concentration was above this level. This, I think, is due to the fact that, although fewer men were employed here, they of necessity worked much closer together than the men at

PERCENTAGE BASOPHILIC AGGREGATION.

Place and date.		Below 0.4%	0.41 to 0.60%	0.61 to 0.90%	0.91 to 1.24%	above 1.24%
WINDSOR July, 1935. 96 men. incomplete survey.	%	47	19	17	3	10
	Men	49	19.9	17.7	3.2	10.2
Sept., 1935. 259 men. complete survey.	Men	112	85	46	9	7
	%	43.3	32.8	17.8	3.5	2.7
June, 1936. 111 men. (lay offs)	Men	38	42	21	5	5
	%	34.3	37.8	18.9	4.5	4.5
TORONTO July, 1935. 67 men.	Men	12	15	18	9	13
	%	18	22.4	27.0	13.3	19.3
Nov., 1935. 60 men.	Men	15	27	9	5	4
	%	25	45	15	8.4	6.6
Feb., 1936. 32 men.	Men	12	12	7	1	0
	%	37.5	37.5	21.9	3.1	0

Windsor, where the factory floor was about 200 yards long and 70 feet wide. Also the men in Toronto for a time refused to wear their safety equipment and increased their hazard. This was partly offset by good ventilation, but a generally higher count was found among these men. Those about 1.24% were not nearly as high as those in the same bracket in the Windsor plant, where the production was four times as high. This naturally greatly increased exposure of those in the immediate vicinity of the discing operation.

A rather striking example of how doing away with the disc-sanding improved conditions is seen by comparing the records of the Toronto Branch for July and subsequently. Disc-sanding was stopped after the July survey had been made. The later records show that the aggregate counts had moved to a substantially lower level and the number of men in the bracket above 1.24% was markedly reduced. Counts from 1.24% to 1.50% or thereabouts do not necessarily indicate any immediate danger, but these men, if exposed much longer, would certainly develop into cases of clinical plumbism. Men removed from exposure when high aggregate counts were noticed, had definite lowering of such, almost immediately. Weekly checks were made and when normal levels (below 0.75%) had been maintained for several weeks, they were returned to their place of work. As has been stated, the bulk of the normals lay below 0.60%; those lying between 0.90% and 1.24% were regarded with suspicion and closely watched. A group of men hired to replace those removed from exposure showed continual increases, though still within normal limits, even with the reduction in the discing operations. One man of this group rose from 4.45% to 1.15% within twenty days! Due to lay-offs and short periods of work for some weeks, he was not recorded again for two months, when he was found to have a count of 3.35% and immediately removed from exposure.

Referring back to the table, a total of eight men were removed from exposure at the Windsor plant between July and September 1935. Two clinical cases were encountered at the beginning of this period. In Toronto during July 1935, of the thirteen men above 1.24%, two were removed. The remainder were just above that level and

became lower with the reduction of the sanding operation. Two more were removed in November, and by February, 1936, there were no counts higher than 0.93%. In May, 1936, a check of all those in Toronto who had at any time seemed suspicious showed that all were well within normal limits, with the exception of one, who was at 0.97%. This was not the same man who had a high count in February.

It is rather interesting to note that we were able to tell, within fifteen feet, where a man worked if his count was up at all. In every case, he was a disc-sander or a nearby workman. It is adequate proof of the reliability of the method. We were also able to tell if a man was a disc-sander who was buffing lead. Emanating from every one of them, as they sat down before you to have their finger jabbed, was a very peculiar sweetish metallic odour. It was distinctive in that it resembled no other odour that I can recall. The solderers, on the whole, were singularly free of increased counts. This, in part, can be attributed to the fact that the solder pots were thermostatically kept just above melting point, and were covered with forced-draught fume hoods.

Theoretical Aspects.

The foregoing account of the McCord method and its interpretations would not be complete without a discussion of the merits and theoretical aspects of the work. As far as the automobile industry is concerned, the clinical plumbism which is encountered is of the rapidly fulminating type, sometimes labelled "acute." McCord's method seems to be more applicable to this than to the chronic type—hence its embracement by the industry. In the chronic case, the counts obtained more closely approximate those by the ordinary methods. The counts here are lowered in proportion to the actual degree of plumbism manifested, so that in such circumstances, McCord's method may prove to be unreliable. This brings forward the interesting speculation that lead at first acts as an irritant to the haemopoetic tissues, and as the amount of lead increases, it later destroys those tissues. McCord maintains in common with others that stippling, reticulation and polychromasia are all manifestations of the same thing, namely, regenerative activity. It is usually taught that stippling is an indication of degeneration, but this seems open to much question. Key maintains that stippling is a manifestation of some change in the basophilic substance after the cells enter the circulation, as it is not present in the bone marrow, and that reticulosis is part of the transition of the cell to maturity. In this, view, Key has been supported by others, but McCord insists that both stippling and reticulation are manifestations of immaturity of the cell. Both are found in the newborn, but I do not know whether both are encountered together in the activity seen after blood loss or commencement of liver therapy in pernicious anaemia. At least, I have seen no remarks on this question. McCord's premise is that stippling must originate while the cell is within the bone marrow. The finding of stippled cells or aggregates in the blood stream does not prove that the change has occurred in the marrow rather than in the blood, as Key would have it. Even though he cannot demonstrate stippling in the red cells of the marrow, this does not prove that they do not originate there. McCord's method of aggregation certainly brings out far more than can be seen by the usual methods, but his opponents consider that he has constructed an artificial picture and created things which were not originally present. This is possible and may explain why there is no correlation between the stippled cell counts and basophilic aggregate counts in any set of duplicate smears, though both will satisfactorily demonstrate lead absorption and the approximate degree of toxicity resulting therefrom. If McCord is correct, in that stippling, reticulation and polychromasia are manifestations of the same phenomena and that they can be all demonstrated at once as basophilic aggregates, it would not be wrong to assume that the

aggregate count should equal the total of the stippled cell, reticulocyte and poylchromatic cell counts obtained in any set of duplicate smears. With this consideration in mind, I made duplicates of the February smears of the Toronto men and stained them to demonstrate reticulation stippling and polychromasia as well as aggregation. Unfortunately, I was unable to examine any except the aggregates for several months, and when I came to go over the others, I found that the climatic effects had so distorted and destroyed the smears as to make them useless. I have not been able to make a subsequent attempt, but feel that some information might be gained from such a procedure. It may be a means of further investigation of the early life of the blood cells. As a matter of speculation, it may be suggested that McCord's method alone cannot be completely satisfactory, but that in all probability, if used in conjunction with the ordinary methods, much accurate diagnostic information will be made available. His technique is certainly by far the simplest, speediest and easiest to read.

In conclusion, I should like to express my sincere appreciation to Dr. C. A. Langmaid, of Windsor, for his generous assistance in many ways, and also to Mr. H. W. Wakefield, of the Department of Medicine, Toronto General Hospital, for his helpful suggestions and encouragement.

THE LEUKAEMIAS

By W. I. BRESLIN, '38.

Leukaemia is a morbid condition of unknown origin, widespread throughout the body and characterized by a hyperplasia of the leukopoietic tissues with qualitative and quantitative changes in the white blood cells.

Age Incidence—*Acute*: In the first five years of life, the lymphatic type is seen; up to 25, the myeloid. The monocytic form may occur at any age. *Chronic*: This condition is rare in childhood. The myeloid type is commonest from 35 to 45 years; the lymphatic from 45 to 60 years.

Etiology.—This is obscure. Acute forms suggest the picture of an inflammatory process, the chronic rather one of new growth involving the leukopoietic tissues.

Classification.—This is based upon the type of white blood cell chiefly involved in the lesion. When neutrophil, the leukaemia is spoken of as myeloid or myelogenous, when lymphocyte as lymphoid or lymphatic and so forth.

CHRONIC LEUKAEMIA—SYMPTOMS.

These are very insidious in nature. The patient may look well or extremely ill. In chronic lymphatic leukaemia, there is usually an antecedent swelling of the lypmhatic glands or tonsils, in chronic myeloid enlargement of the spleen is usually a prominent feature.

1. *Associated with Anaemia.*—Weakness, fatiguability, breathlessness, cardiac dilatation and decompensation with dyspnoae, gravity oedema, accumulation of transudate in the serous cavities, pericarditis, pleurisy.

2. *Pressure Symptoms.*—(a) Spleen (myeloid)—there are all grades of enlargements which may be painless or may simulate an abdominal emergency (infarction, perisplenitis and friction rub). The size may vary during the course of the disease. Size is not necessarily associated with total white blood cells, but rather with increased blood volume.

(b) Lymph glands (lymphatic).—These glands are discrete, firm, but not tender. All the accessible superficial glands may be involved. Occasionally abdominal lymph glands are felt. Sometimes only the deep glands enlarge, e.g., mediastinal, giving early pressure symp-

toms. Rarely do the glands attain the size of those in Hodgkins of Lymphosarcoma. Mikulicz Syndrome — salivary, lachrymal glands are enlarged. Hearing and vision are impaired. There is exophthalmos and a dry mouth and throat. Terminally in myeloid leukaemia, the glands will enlarge or the spleen in the lymphatic type.

(c) Liver.—Usually palpable, firm, smooth. There may be associated gastro-intestinal symptoms, such as anorexia, diarrhoea, and haemorrhages. Lymphatic leukaemia may cause gastric obstruction resembling carcinoma.

3. *Metabolic Disturbances.*—There is marked weight loss and cachexia. B.M.R. is elevated in most untreated cases. Oxygen consumption is increased, due to over-active tissues producing and destroying W.B.C., and also because of the increase in the number of cells. Sturgis claims that the increased B.M.R. is roughly proportional to the severity in myeloid leukaemia. Following radiation, there is a brief rise, followed by a fall to normal, lasting three to six months in favourable cases. There is no such parallelism seen in lymphatic leukaemia.

Attention may be directed to: 1. The Nervous System—which may be involved from the cortex down to the peripheral nerves, particularly cranial VI. and VII. Haemorrhages occur into the retina and labyrinth, giving a Ménière-like condition. True optic neuritis with markedly impaired vision is rare.

2. Genito-Urinary System. — There may be haematuria and priapism (in the myeloid type). Both give menstrual disturbances, while prostatic enlargement is seen in the lymphatic type. Bence Jones' bodies may be found in the urine.

3. Pulmonary System. — Cough is present, due to bronchitis. There may be fever, pulmonary congestion and oedema simulating tuberculosis.

4. Skin, Bones, Joints. — Petechiae occur, but are uncommon early. Later there are purpuric manifestations, with even large haemorrhages into pleura, peritoneum and mucous membranes. These are not usually fatal. Skin infiltration is more frequent in lymphatic leukaemia, e.g., tumors and hematomata, which are made up of cells which are even more immature than those seen in the bloodstream. Herpes Zoster is not as commonly present as in Hodgkin's disease. Pain over the long bones simulating osteomyelitis is not common. There is often pain over the gladiolus opposite fifth costal cartilage in myeloid leukaemia and this pain is aggravated by pressure. X-ray may show osteosclerosis, periosteal lifting, moth-eaten medulla; in children, bony changes are just proximal to the metaphysis.

BLOOD.

The blood is thick with high volume, especially in myeloid leukaemia. In the early stages, anaemia is absent or slight. Later it is more severe and normocytic. At times, however, it simulates pernicious anaemia. Anisocytosis is marked only in later changes. Polychromasia, stippling, reticulocytosis, and normoblasts occur, the latter even becoming more numerous than in pernicious anaemia. All these characteristics are uncommon in lymphatic leukaemia.

The platelets in myeloid are usually increased and their numbers fluctuate. If they are persistently low and unchanged by remissions, it is a bad sign. In lymphatic leukaemia, they are decreased in accordance with bone marrow insufficiency due to lymphatic infiltration.

MYELOID.

W.B.C. 100,000—1,000,000.
Predominant cell is the polymorph.
Premyelocytes and myelocytes, 25-50%.
Granulocytes, 90-95%.
Mature basophils and eosinophils, 10%.
Myeloblasts, 1-3%.
Lymphocytes relatively decreased, but actually increased.
Naegli claims that blood in leukaemia is only quantitatively different from normal bone marrow.
Peroxidase stain positive.
Indo phenol positive.
Supra—vital staining is helpful.

LYMPHATIC.

Rarely as high.
Lymphocytes, 90-99%.

Lymphocytes vary in size and appearance; are unlike normal, most are small with a rim or no apparent cytoplasm, scanty azure granules, poorly staining nuclei.
Rieder cells (lobulated) appear.
Smudge cells, young, non-resistant cells. There are some with a great deal of cytoplasm, with rounded knob like buds at the periphery.
Lymphoblasts infrequent. Plasma cells are present. Few polymorphs. Eosinophils and basophils rare.

Coagulation time in both is prolonged. Uric acid, Blood Calcium and N.P.N. are often high. Intercurrent infection alters both of these pictures.

ACUTE LEUKAEMIAS.

These are characterized by sudden onset, fever, marked prostration and a rapidly progressive fatal outcome. There is a tendency to necrotic processes in mucous membranes and throat. The platelets are reduced and haemorrhages from the mucous membranes and skin occur. Anaemia is early and severe. Polychromasia, stippling, anisocytosis are present, but nucleated reds are rare. Despite the fever, there is pallor. Bone and joint pains are present, and the spleen, liver and lymphatic glands are enlarged, but not as markedly as in chronic leukaemia.

1. *Myeloblastic.* — The white blood count ranges from 25,000 to 30,000. At times, it is as low as 400, although leukopenia is rare at the start of the disease. Myeloblasts reach 60%. These are large and mononuclear, with round oval nuclei, deep blue cytoplasm, no granules, and with a fine nuclear chromatin containing four or five nucleoli. Premyelocytes are present and also myelocytes. Mature polymorphs are scanty and basophils and eosinophils are rare.

2. *Lymphoblastic.*—The initial count is often 10,000. Again we see a large mononuclear, but with coarse stippled chromatin, a dense nuclear membrane with a clear peri-nuclear zone and 1-2 nucleoli. Very occasionally there are azure granules. There are very few polymorphs and still fewer of the other granulocytes. Occasionally there are up to 10% myelocytes. There are no premyelocytes or myeloblasts, however.

3. *Monoblastic.* — The most typical case was described by Clough.

(a) A true monoblast-nucleus has fine chromatin and several small nucleoli. There is a basophilic cytoplasm and no granules (Schilling type).

(b) Mononuclear with grayish-blue cytoplasm containing fine dust-like lilac granules. Nucleus is bean shaped and of fine reticular chromatin.

(c) Larger cells with irregular cell boundaries, pseudopodia and a folded-looking nucleus. There are some fine granules and a foamy cytoplasm. The rest of the cells are polymorphs, a few myelocytes and myeloblasts. Plasma cells can be seen. (Naegle type of Monoleukaemia.)

UNUSUAL TYPES OF LEUKAEMIA.

1. Eosenophilic. — Chiefly mature eosinophils 25 to 90%. Myelocytes, 2 to 8%. There is a moderate anaemia and the spleen is enlarged.

2. Neutrophilic.—Few or no immature cells.

3. Plasma cell leukaemia.

4. Megakaryocytic.—Some cases of aleukemic myelosis show many of these cells.

5. Chloroma.—Chloro-leuko-sarcoma. These are greenish tumors, seen in the periosteum, ligamentous structures of skull, orbits, sinuses spine, ribs, sacrum. At times they manifest themselves as skin tumors. Cranial nerve palsies are frequent. Blood picture is like that of acute myeloid leukaemia, especially the aleukemic phase.

6. Aleukemic Leukaemia. — Seen in all leukaemias, especially acute leukaemia and particularly lymphatic. Normal white blood count or leukopenia is present, so changes are qualitative rather than quantitative. There may be a few nucleated reds and other immature red blood cells. The anaemia is much more severe than in chronic leukaemia. Haemorrhagic phenomena are common.

Complications:

1. Haemorrhagic accidents.
2. Progressive anaemia.
3. Myocardial insufficiency.
4. Tuberculosis and other infections may flare up.

Prognosis. — Chronic Leukaemia: Death in one to four years, occasionally 15 to 16 years. Remissions occur spontaneously or due to therapy. The acute forms are rapidly fatal.

Diagnosis.—The difficulty is largely in the atypical forms, e.g., the patient is seen for the first time with aleukaemia lymphatic leukaemia, marked leucopenia, total granulocytopenia, severe anaemia, decreased platelets, and haemorrhagic phenomena. The following is a list of conditions that may be confused with leukaemia and some characteristics that may help differentiate them.

1. Aplastic Anaemia.—This is never a normoblastic or hyperchromic anaemia. There is no normoblastic regeneration, much lower platelet count and poor clot retraction. Lymphocytosis is relative and white blood counts are decreased. No physical changes are present (spleen, glands, etc.).

2. Acute Benign Lymphadenosis.—Both this and acute lymphatic leukaemia are diseases of children and young adults (laboratory workers). There is high fever, adenopathy, enlarged spleen, sore throat and at times ulceration. There is relative and absolute lymphocytosis. There are no red blood count changes, no platelet reduction, no haemorrhagic phenomena, the patient always recovers. Immature white blood counts are uncommon, but when present, are numerous with different types present at the same time. Heterophile reaction is frequent, positive and if so is diagnostic, occurring in no other condition except allergic phenomena.

3. Occasional cases of Granulocytopenia.—There is fever, necrotizing angina, ulceration of the mucous membranes, platelet reduction and haemorrhage. The latter is never as pronounced as in leukaemia. Regional glands are but slightly enlarged. Patient is pale, but mucous membranes are of good colour. Immature white blood counts are uncommon except terminally or with recovery.

4. Purpura.—There is a history of previous attacks. The white blood counts are normal. Anaemia is secondary and only after repeated haemorrhages. Platelet count is low. Bleeding time is prolonged, and clot retraction does not occur.

5. Resemblance of Aleukaemic Leukaemia and Hodgkin's.—Hodgkin's disease involves the posterior cervical lymph glands with late generalization. The masses are larger, pressure symptoms are more common and the Gordon Test is positive. The Pel Epstein type of fever is common in Hodgkin's. In lympho - sarcoma, there are larger masses, but glands are indistinct, and liver and spleen are rarely enlarged. Occasionally a leukaemic blood picture is engrafted on lympho - sarcoma. In neither Hodgkin's nor lympho - sarcoma are there skin, pharyngeal or laryngeal infiltrations as in leukaemia.

6. Pernicious Anaemia.—Both it and leukaemia (at times) are hyperchromic. In pernicious anaemia, there is a leucopenia and relative lymphocytosis. Occasional myelocytes are present as are nucleated reds. Platelets are decreased, spleen is occasionally enlarged, thus imitating chronic myeloid in the aleukaemic state. Note reticulocyte response to liver and presence of achyllia gastrica.

7. Miscellaneous — septicaemia military T.B., subacute bacterial endocarditis, thyphoid. Aleukaemic leukaemia may simulate splenic anaemia, hemolytic icterus, thrombosis of splenic vein, Gaucher's disease. Even pneumonia, with myelocytes may cause confusion.

8. Leukaemoid States, e.g., Anaemia pseudo leukaemia enfantum, Von Jaksch's anaemia.

9. Infectious Mono nucleosis. — Usually in children, with sore throat, high fever, cervical gland enlargement. Liver and spleen are enlarged and tender in many cases. White blood count reaches 15,000 to 20,000, 75% to 90% mono-

nuclear of a lymphoblastic type. In adults, a rash may be an early sign.

So we stress in blood picture quality rather than quantity. Failing this biopsy, lymph glands of bone marrow, and often autopsy findings are necessary for diagnosis. Even at this stage, there is difficulty in distinguishing the leukaemoid state and true leukaemia. Autopsy material treated with alcohol, benzidine and peroxide, shows deep blue colour at once in cases of chloroma, slow and less blue in myeloid and no change in lymphatic leukaemias.

Pathology:

Myeloid. — Bone marrow of long bones is changed from fatty to active marrow and is grey in colour. At times may be red or even purulent, crowded with myeloblasts. Red blood count activity is marked (in advanced stages, little erythroblast tissue is present and megakaryocytes are abundant.

Spleen. — Enlarged, firm and cuts with difficulty: Haemorrhagic infarction filled with cells of the myeloid series; Malpighian follicles not seen; lymph nodes, liver (fatty degeneration), heart, lungs and blood vessels crowded with myeloid cells).

Lymphatic. — Glands packed with lymphocytes, wiping out normal architecture; spleen is the same and shows infarction. The liver is enlarged with collections of perivascular lymphocytic nodules seen also in gastro-intestinal tract, brain, kidneys, and spinal cord. Bone marrow shows replacement of erythroblastic and myeloblastic tissues by lymphocytes.

Treatment:

(a) General measures, diet, rest, etc.

(b) Transfusion as a temporary measure.

(c) Arsenic up to toxic doses especially in chronic myeloid. There is danger of poisoning to the cumulative effect.

(d) Benzol in capsules with olive oil, 0.5 gms. daily in four doses. Maximum of 4 to 5 gms. in seven to ten days (most valuable in myeloid). Has toxic effect on red blood count. Platelets first rise and then fall. Toxic systemic manifestations are headache, vertigo, bladder irritability, renal congestion, impotence and gastric disturbances.

(e) Radiation—done over spleen and over glands causing pressure.

Radiation has no necrotizing effect, but stimulates the premature cells to ripen. It is probably not good, therefore, in acute leukaemia. If successful, there is a temporary exacerbation whence B.M.R. goes down and Haemoglobin and red blood counts rise.

The white blood count falls and immature forms tend to disappear. There is a more or less parallel improvement in liver, spleen and glands. One should attempt to get the white blood count to 30,000, counting 24 hours after radiation. The best guide is the patient's condition. Contra indications are aggravation of general symptoms, too rapid a fall in white blood count, increase of premature white cells and increased anaemia.

(f) Radiation plus iron and transfusion as required.

DIAL "CIBA"

Dial calms excited, irritated nerves, and for such occasions as nervous insomnia, mental and traumatic agitation, pre-operative restlessness, etc., it will fulfil all the requirements of a good hypnotic.

Cibalgine "Ciba"

Cibalgine represents a non-narcotic analgesic and antipyretic worthy of the physician's confidence. It is indicated in the treatment of pain of every description, febrile manifestations, nervous excitement, insomnia due to pain, dysmenorrhoea, etc.

Ciba Company Limited
MONTREAL

THE FOETAL SKULL

By CLEMENT McCULLOCH, '39.

The development of the foetal skull will be traced through its three principal stages. These are membranous, cartilaginous and bony, in order of appearance. An effort will be made to group the changes studied, so they may be easily understood. Nearly all detail will be left out.

While tracing the development of the skull, it is important to remember that the structures considered lie in relation to the vertebral column, the brain and part of the digestive and respiratory systems. Therefore, the changes to be studied are concurrent with changes in, and modified by, these neighbouring structures.

The Membranous Skull.

The membranous skull is a structure which reaches its height of development in the second foetal month. It is formed of a paraxial part around the brain, and a visceral part around the mouth and nose region.

The paraxial part appears first around the anterior end of the notochord, below the midbrain, as a dense plate of embryonic connective tissue. At the caudal end of this plate four segments are seen. These are considered to represent the segments of the hypoglossal nerve. The plate is enlarged to the sides and forward, so that it eventually completely surrounds the developing brain. From this structure all the mesodermal structures of the region, membranes of the brain, bones and cartilages, connective tissue and nucleus, will be developed.

The visceral part forms below the paraxial membranous skull. Here embryonic connective tissue forms a ring around the anterior end of the digestive tract. In certain places, called gill slits, the connective tissue is deficient and the ectoderm, or skin, comes in contact with the endoderm of the gut. The connective tissue between these slits forms the gill arches. This part of the membranous skull forms the face region.

The Cartilaginous Skull.

The cartilaginous skull appears about the seventh week and reaches its height of development in the third foetal month.

In the paraxial mesenchyme, in the region of the base of the cranium, a plate of cartilage develops at each side of the cranial end of the notochord. These plates grow and fuse dorsal and ventral to the spinal medulla. Two more cartilages form in front of the notochord. These anterior and posterior sets of cartilages (prechordal and parachordal plates) then fuse to form one plate. The prechordal plates fuse around a diverticulum from the stomodeum (Meckel's) to form the floor of the hypophyseal fossa. From each side of the prechordal plates there grows out a small wing anteriorly, and a great wing with the lateral pterygoid lamina posteriorly. The fused anterior end of the prechordal plates forms the ethmoid plate. A medial and two lateral processes develop from the ethmoid plate. The nasal septum, the perpendicular plate of the ethmoid and the crista galli develop from the medial part. The ethmoidal labyrinths and the altar cartilages of the nose develop from the sides.

In the region of the side of the cranium, the ectodermal auditory vesicle is formed. Around this is collected a thick layer of embryonic connective tissue, forming the membranous otic capsule. In this, cartilage forms, starting on the lateral side. This cartilage surrounds the semicircular canals, and then the cochlea, forming the cartilaginous otic capsule. By this means, two cartilaginous, joined, masses are formed. The ganglia of the facial and auditory nerves lie on the antero-superior aspect between these masses.

The facial nerve passes to its nucleus in this situation, and then over the junction between the two masses and downwards posterior to any structures of the first and second branchial arches. The cartilaginous otic capsule fuses with the lateral side of the parachordal plate around the ninth, tenth and eleventh cranial nerves. From this fused mass are formed the petrous and mastoid bones.

In the region of the dome of the cranium, cartilages are formed in the embryonic connective tissue. These are called tecta and form a bridge over the brain, joining the two occipito-temporal regions.

In the visceral mesenchyme cartilages also appear. They are parts of the embryonic gill arches. At the cranial end of the first arch the incus is formed separately. Ventral to the incus the malleus is at first continuous with, but later separates from, Meckel's cartilage. Meckel's cartilage forms the rest of the first, or mandibular arch. The second arch lies between the tympanic bone and the facial nerve. The dorsal extremity forms the incus. The rest of the arch will form the tympanohyoid and stylohyoid bones, the stylohyoid ligament, the lesser cornu and part of the body of the hyoid bone. The third arch forms the greater cornu and body of the hyoid bone. The fourth and fifth arches form the thyroid, and the sixth the corniculate and arytaenoid cartilages.

The Bony Skull.

The bony skull is a structure that reaches its height of development after birth. The first bony point to appear is in the mandible during the fifth week of foetal life. The bony skull is developed in two ways:

1. Bone is laid down by ossification in the cartilage of the chondrocranium, cartilaginous bone.

2. Bone is laid down by the embryonic connective tissue surrounding the chondrocranium, membrane bone. The cartilage that is deep to this bone is usually absorbed.

The cartilaginous bones of the skull are:

1. Paraxial Region.

All the occipital except the posterior part of its squama.

The bodies of the sphenoid and ethmoid.

The perpendicular plate and crista galli medially, the ethmoidal cells and conchae laterally, the cribiform plate between, in the nose region.

The sphenoidal wings, lateral pterygoid plates and hamuli of the medial pterygoid laminae of the sphenoid bone.

The petrous bone and part of the mastoid bone.

2. Visceral Region.

Part of the malleus, incus and mental ossicles.

Some of the tegmen tympani.

The stapes, styloid process and hyoid bone.

The membrane bones of the skull are:

1. Paraxial Region.

Posterior part of the occipital squama.

Parietals, frontals, nasals.

Squama of temporal bone and part of the mastoid process.

Tympanic bone.

2. Visceral Region.

Laterally the mandible, maxillae, lacrimals, zygomatic, alatemporalis of the sphenoid and membranous part of the malleus

Medially the premaxillae, vomer, palatines, medial pterygoid plates.

Some examples of membranous bones covering cartilage are:

1. Paraxial:

Over the tecta in the vault of the cranium; the squamosal, forming part of the mastoid and the lateral wall of the middle ear.

The nasal bones covering the nasal capsule.

2. Visceral:

The mandible surrounding Meckel's cartilage.

The vomer forming on each side of the ethmoidal nasal septum.

The lacrimal forming over the cartilage of the nose capsule.

Ossification of the membrane bones

begins during the second foetal month, except for the lacrimal and tympanic bones which appear during the third foetal month.

Ossification of cartilage bones is much more complicated than that of membrane bones. Cartilage bones ossify by several centers some of which join with developing membrane bones. Cartilaginous tend to develop later than membrane bones, between the end of the second month and birth.

The occipital and sphenoid appear during the second and third month; the occipital and great and small wing of the sphenoid during the second month; the other parts during the third month.

Cartilage bones of the lateral nasal wall appear in the fifth foetal month, those of the medial nasal wall a year after birth.

The petrous bone and auditory ossicles appear in the fifth foetal month, the tympanohyal and mental ossicles just before birth, any other gill arch structures after birth.

The parts of the occipital and temporal bones fuse after birth, those of the sphenoid both before and after. The occipital, sphenoids and ethmoids are fused in their basilar parts by the twenty-fifth year.

The skull at Birth.

The skull at birth is relatively large in comparison with the size of the body. The size of the cranium to that of the face is 8:1. In the adult this ratio is 2½:1. The face is small due to the fact that the teeth are not erupted and the air sinuses take up little room; the orbits are close to the palate and the piriform aperture is circular and lies between the orbits, not below them. The zygomatic arch is at the level of the superior alveoler process and the perpendicular heights of the maxilla, palatine and ethmoid bones are small. The size of the face is also associated with a small mandible. The teeth present are not erupted, the alveolar processes are poorly developed, the angle is obtuse, and the coronoid process is the highest point of the mandible. The condyloid process lies more posteriorly in a shallow mandibular fossa.

The sutures are of particular interest. They are formed by parts of the membranous or cartilaginous skull and join together the bony elements. The edges of the bones are rounded and do not interlock. Therefore the head can be moulded, one bone passing over another. The skull is split from the occipital bone to the tip of the jaw by a suture. The width of this suture is increased in three places, forming an occipital fontanelle at the occipital squama, a sagittal fontanelle between the parietals, and a frontal fontanelle between the frontal and parietal bones. The frontal fontanelle has a long anerior limb lying between the frontal bones. It is usually the last fontanelle to close—during the second year. The occipital fontanelle is Y shaped, the vertical limb of the Y extending towards the face. Four other fontanelle are formed at the lateral ends of the anterior and posterior borders of the parietal bones. The ears and orbits lie close to these.

The size and shape of the skull at birth have been carefully studied. The head is ovoid, narrow in front and wide behind The widest point is at the parietal eminence and measures 9½ cms. The occipito-mental measurement is 13 cm. and the occipito-frontal is 11 cms. The frontal eminences stand out, there being no superciliary arches and the orbits are large.

Three places where development is not complete should be noticed.

1. The mastoid process has not developed, and the stylomastoid foramen looks laterally.

2. The external acoustic meatus is cartilaginous, the tympanic bone being small.

3. The cribiform plate is not yet developed, the upper part of the nasal cavity is narrow and only separated by membrane from the brain.

FEATURE SECTION

Notes of a Student—One Hundred Years Ago.

(Through the kindness of Dr. E. Stanley Ryerson, Assistant Dean and Secretary, Faculty of Medicine, University of Toronto).

At the open meeting of the Medical Society on November 24th, in the address on "The Medical Student, Past, Present and Future," some extracts from the notes of Dr. John Beatty of Cobourg, taken during his medical course at The College of Physicians and Surgeons of New York (later Columbia) in 1831 to 1833, were read by the lecturer, Dr. E. Stanley Ryerson, a grandson of Dr. Beatty.

In the notes from the lectures on Surgery of Dr. Alexander H. Stevens (1789-1869), a famous New York surgeon of this period, who later became President of the American Medical Association and one of the co-founders of the New York Academy of Medicine, the following selections appear:

November 15th, 1832.

Nature of inflammation. — Hippocrates supposed it to consist in an increased flow of blood to any part. Erasistratus supposed it to consist of an escape of blood from its proper vessels. Sydenham supposed that some chemical change took place by which the materials of blood acted one on another. Boerhaave—attributed it to mechanical causes arising from an error loci of the globules of blood. Spissation of the blood also allowed as a cause by him and he attributed the increased heat to the greater degree of attraction between the globules. Objection—Mechanical. Cullen maintained that the first condition of the capillaries in inflammation was spasm, to overcome which the action of the heat was increased by the "vis medicatrix naturae." Irregularity of circulation is an effect—but not a cause of inflammation. Brown held that inflammation was an effect either of an increase or decrease of stimulus in the vessels of the part affected. Hunter defines it to be an active dilation of the parts.

Friday, November 16th, 1832.

Effects of inflammation on various structures and the appearances. Of cellular membrane—effusion of coagulating lymph—secretion of pus, etc. Fibrous structure loses its bright color and strength, becomes thick and coated with adhesive purulent matter—excessive pain. Tendons—seldom swell—lose their shining appearance—given no pain—tend to sloughing. Ligaments swell, lose their strength—become dark colored—and after severe inflammation become like jelly. Periosteum thickens—arising from deposition of co(agulating) lymph—just afterwards of pus—very severe pain. Cornea—Muddings — slow in its progress. Absorbents—extends along their course—enlargement of the glands in which they terminate—extreme pain in a depending position. Arteries — coats thickened — friable—loss of transparency of inner coat—terminates sometimes in aneurism—or in deposition of osseous matter. Veins — Nerves—excessive pain radiating along the course of the nerve—pain most acute, with a peculiar burning sensation. Mucous membrane—bright red, thickened, easily torn production of pus—sometimes false membrane.

. . . Serous membranes—thickened—lose transparency, increase of exudation—sometimes lymph-Tunica arachnoides becomes thickened — bloated with water. Brain—2 kinds . . . Bone-pain very severe—Intervertebral substance swells and becomes soft—Synovial membrane ends in increase of secretion. Cartilages . . . Liver for most part softened—more friable than natural—sometimes hardened.

December 7th, 1832.

Burns—Distinguished by the depth and extent of the injury—Superficial wounds most painful—attended with

slight redness—ends in desquamation of the cuticle—alleviated by cold applications. Second kind—skin blisters limpid serum—pain very severe—continuing two or three days—cuticle falls and is thrown off and new one formed. Sometimes in these cases cuticle rolls up and immediately exposes the true skin. Third—cuticle destroyed—if this be the case the pain is most excruciatingly severe—owing to the exposure of the nerves. Fourth—The whole of the skin is destroyed—5th degree—skin and some parts beneath it are destroyed—in this case the pain may not, indeed is not—so severe as when the cuticle alone is destroyed. Most severe kind—limb is charred through—attended with very little complaint of pain. Burns with eschars in general are deeper than they appear to be on first appearance.

Constitutional symptoms of series —acute pain is first felt—this may be so severe as to kill—if does not— patient falls into stupor of mind and body —which may last two or three days. If the patient survives these symptoms are followed by congestive fever—if the patient then lives to ninth or tenth day, will have escaped the danger of this affection. But many die of suppurative fever. Danger arises from extent of the injury. First from the shock of nervous system—second, congestive fever—third, suppurative process.

In the worst cases of burns there is no complaint of pain. Long continued coldness—nausea— vomiting — convulsions are dangerous symptoms. If the lower lamina of skin be saved there will be no deformity by contraction.

January 9th, 1833.

Showing Pictures ! to himself. (In the notes in Surgery from lectures by Dr. Valentine Mott (1785-1865).)

About Dr. Mott it was said by Gross that no surgeon ever tied so many vessels for aneurysm. It is recorded that when thirty-three years old he ligated the innominate artery two inches from the heart and the patient lived twenty-eight days. He performed Lithotomy 165 times. The notes of his description of this operation and other topics were as follows:

February 18th, 1833.

In the operation of Lithotomy the accelerator and levator ani muscles are cut, the former, however, very slightly —if pushed up a little. The arteries cut are the transversus perinei and the artery of the bulb, but they are small. If the pudica interna be cut it is in consequence of a mal operation. Difficulties met with—First, something may cause the knife to slip from the staff—or give it a wrong direction, by which some part other than wished may be cut—try again. Second, size of stone —may enlarge the incision or try to break down the stone—many of which are of such a nature as to crush very easily—also when very small they will be troublesome. When the internal pudic artery is cut there is much difficulty in restraining the hemorrhage. Average success. Consequent treatment—will generally be antiphlogistic —if symptoms of inflammation of bladder or peritoneum occur—must treat actively and appropriately. About the twelfth to fourteenth day the urine will begin to pass through the penis this is sometimes accompanied with the most severe rigors and chills—owing to the irritation of the unine passing over the inflamed surface of the urethra. Treat by giving anodynes in free doses.

Sometimes secondary hemorrhage apparently of a venous character occurs two or three weeks after the operation—probably from congested veins about cervix vesicae.

February 20th, 1833.

Relative Anatomy of the Popliteal Artery—becomes so as soon as it passes through the triceps muscle. In this space the artery lies deepest and nearest the inner condyle of the humerus. Popliteal vein more superficial and external. Nerve still more external and most superficial of all. Aneurysms occur very frequently in this part of the

artery and from its nearness to the bone 'tis apt to injure the bone.

Knee-joint composed of the Femur, the Tibia and the Patella. Upjohn was laughing ha! ha! (the slip of writing triceps for quadriceps and humerus for femur is a common one with students).

Monday, November 12th, 1832.

DR. J. M. SMITH'S LECTURES ON THE THEORY AND PRACTICE OF PHYSIC PLAN OF LECTURES ON THEORY AND PRACTICE.

Two Divisions—1st Institutes and 2nd Practice of Medicine. Institutes arranged under 4 heads.

1st Physiology—Hygiene—Pathology—Therapeutics.

First Physiology—Notice — Properties—Functions—Peculiarities of human race, viz., those ensuing from age—condition — sex—temperaments — different tribes and races of men—Relations of man with external things as these agents affect us Physically 2nd in our moral attributes.

2nd Hygiene—relating to preservation of health and prolongation of life—Including the history and influence of the Nonnaturals—Air Aliment Exercise—Passions—Retention and Excretion. Sleep and waking.

3rd Pathology—We shall inquire what disease is.

Div — Etiology—Symptomatology—Semeiology.

The term "*cause*" in medicine is used in a very vague and indeterminate sense.

In connection with Pathology consider the three modes in which diseases terminate, viz., Health—Death—In other diseases.

4th Therapeutics.

Practice of Medicine.

First notice Classification of Diseases.

Sauvage made first classification—Linnaeus—Boyd—Cullen.

Plan pursued—Divide diseases into Acute and Chronic.

Wednesday, November 23rd, 1831.

Persons should never eat so much as to produce drowsiness. Students especially should avoid excess in this respect, as it impairs the energy of the brain. Drink—The best under almost all circumstances of Health is water. If it were more generally used, there would be less disease by nearly 2/3. Tea and Coffee when used very strong are injurious, more especially to those of a Melancholic Temperament.

Monday, November 28th, 1831.

Of the three grand divisions of the Institutes. Pathology by which is understood that Science which teaches the general doctrines of disease.

There have been many definitions of disease but best is: That it is a certain state of the body in which the functions proper to life and health are not performed or at least imperfectly.

Saturday, December 3rd, 1831.

Therapeutics or the treatment of disease.

There are two general methods of treating diseases. First actively or those means by which we attempt to control the disease. Second. The expectant, in which we wait for the salutary effects of the vis medicatrix naturae. Both these methods are worthy of particular attention, as some diseases require the active, others the expectant mode of treatment.

The intentions in the treatment of disease are:

First, to remove the Cause of the disease.

Second, to remove the disease itself.

Third, to remove the effect.

Medicines are Positive or Negative.

In the indications for cure two objects must be kept in mind. First, those circumstances relating to the peculiar Idiosyncracy of the patient, and second, those relating to the general character or history of the disease.

The course of thirty-seven lectures delivered by Dr. A. H. Stevens from November 13th, 1832, to January 21st, 1833, covered the following subjects: Inflammation, Abscess, Mortification and Gangrene, Erysipelas, Burns, Ulcers, Syphilis, Diseases of Cellular Tissue, Furuncle, Anthrax, Scrofula, Tumors, Tubercle, Carcinoma, Polypi,

Injuries of external violence, Wounds, Haemorrhage, Hydrophobia, Tetanus, Gun-shot Wounds, Head injuries (concussion, compression, Compound fracture of skull).

New York, January 9th, 1833.

Extract from letter from Dr. Beatty to his father:

Revd. Jno. Beatty,
York,
Upper Canada.

Dear Father:

. . . As to the amount I shall want to clear my way and provide the things I want to bring with me, I hardly know what to say. As my last will inform you, I am out of money—If I graduate the fee for it is $25 for "Graduation Fee" and there will be some other incidental expenses attending it—My expenses up will I presume be rather more than it generally has cost me owing to the time of year in which I expect to go.

I have not been able from want of time more than anything else—to ascertain what amount of medicines I had better take up. I think, Father, if you call on some of your medical friends they will be able to tell you better than I can of this matter. You can send as you see fit and I can accommodate my purchases to the amount sent. . . .

Your affectionate son,
Jno. Beatty.

A Short Visit to Hudson Stuck Memorial Hospital, Fort Yukon, Alaska.

By J. Wallace McNichol, '37.

This summer, it was my privilege and rare opportunity to visit for a short few hours one of the most northerly hospitals in the world, where both medical and surgical treatment are available. This hospital is the Hudson Stuck Memorial Hospital, which is operated by the Episcopal Church of the United States, and under the guidance of Dr. Grafton C. Burke. This visit took place on a hot summer's day (July 12th), which made it very difficult for one to realize we were above the Arctic Circle, with a temperature of 85 deg. F.

Fort Yukon is situated on the original site of the Hudson's Bay Company fort, built in 1847 by Alexander Hunter Murray, on the most northerly bend of the mighty Yukon River within the Arctic Circle. This mighty river, which has its head waters 2,000 feet above its mouth in Yukon Territory, Canada, above Whitehorse, flows northwest across Yukon Territory into Alaska until it reaches Fort Yukon, and then turns south-west and finally empties into Behring Sea, a little over 2,000 miles distant. About 80% of the river is navigable, with the Mississippi type of wood-burning, flat-bottomed, stern-paddlers, which shoot three rapids coming down-stream, and continually buck on the average a 6-mile-per-hour current going up. Fort Yukon lies some 360 miles downstream (yet north) from Dawson City, Y.T., and in the days of the goldrush of '98, and still to-day, the river is the main means of travel to and from this famous placer gold city. It is only navigable about three or four months of the year. The variation of temperature is very great, ranging from about 90 °F. above to a record of 76 °F. below zero, two years ago.

Dr. Burke is a man in his fifties. He has been the medical missionary for over thirty years in this territory, and is held in very high regard. He is a man of very great interest, kind and gentle to all those who come under his care. He is very mechanically-minded, which is very fortunate, as frequently circumstances demand he make out of rough material many varied appliances to carry on his work. He is assisted by his wife and two trained nurses.

The present hospital is a very modern log structure of two stories, and over a hundred feet long. It was erected a few years ago to the memory of the late Archdeacon Hudson Stuck, Dr. Burke's predecessor, who was well known over the whole continent, not only by his writings and his great work,

but as the leader of the first party to scale Mount McKinley, highest peak on the North American continent. The hospital has only two private rooms, the rest being semi-private and public. The operating room is remarkably well equipped for almost any type of operation. Dr. Burke has only his scrub-nurse acting as his assistant, and indeed it was most remarkable to learn that he successfully does such operations as cholecystectomies, mastoidectomies and hysterectomies, with so little assistance. The dispensary is very complete. Dr. Burke is a student, many recent texts and pieces of literature were in evidence in his office. As closely as I can recall, there were about 60 beds in all under his care.

The hospital is surprisingly independent. They grow practically all their own vegetables (which are very fine indeed, with nearly four months of continuous day). They make and forge many of their own appliances used in and around the hospital, as well as supply their own electricity. The buildings are heated by burning wood, requiring about 100 cords in a winter for heating and electricity. Transportation costs for bringing in articles is almost prohibitive, ranging from 100% to 300% of cost of original article.

One will be surprised to know, no doubt, that there are no Eskimo at Fort Yukon, the native population being Indian, with many half-breeds. On travelling completely across Alaska, I only saw Eskimo at Beaver, a small settlement 125 miles down stream from Fort Yukon, where a small number have migrated. I was very interested in learning that Eskimo only live along the coast lying north of the mouth of the Yukon River, and as far as 50 to 75 miles inland; beyond that point, they are all Indian.

The population of Fort Yukon is 600 natives and 300 whites. The Indian village there is the largest on the river, and, due to education and treatment by Dr. Burke and his predecessor, Archdeacon Stuck, it is the healthiest village in the area. It is one of the few places among the Indians where the birth rate exceeds the death rate.

The hospital serves the greater part of the northern interior of Alaska. Patients are brought by boat in summer, dog-team in winter, and frequently by airplane throughout the whole year. Dr. Burke has probably more tuberculosis to deal with than any other single disease. At the time I was there, he had a number of small native children with tuberculous adenitis, on some of whom he had already done radical resections. It was of great interest to learn that it is only very rarely that he encounters cancer in any form.

"IS THIS PRODUCT COUNCIL-ACCEPTED?"

This is the first question many physicians ask the detail man, when a new product is presented.

If the detail man answers, "No," the doctor saves time by saying, "Come around again when the Council accepts your product."

If the detail man answers, "Yes," the doctor knows that the composition of the product has been carefully verified, and that members of the Council have scrutinized the label, weighed the evidence, checked the claims, and agreed that the product merits the confidence of the physician. The doctor can ask his own questions, and make his own decision about using the product, but not only has he saved himself a vast amount of time but he has derived the benefit of a fearless, expert, fact-finding body whose sole function is to protect him and his patent.

No one physician, even if he were qualified, could afford to devote so much time and study to every new product. His Council renders this service for him, freely. Nowhere else in the world is there a group that performs the function so ably served by the Council on Pharmacy and Chemistry and the Council on Foods.

Mead Johnson & Company coöperates with both Councils, not because we have to but because we want to. Our detail men can answer you, "Yes," this Mead Product is "Council-Accepted."